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BULLETIN OF THE NEW YORK ACADEMY OF MEDICINE

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BULLETIN OF
THE NEW YORK ACADEMY
OF MEDICINE

MARCH, 1943

ADDRESS OF THE RETIRING PRESIDENT

MALCOLM GOODRIDGE*

FOUR years ago on the occasion of my first presidential address I confessed to a feeling of my inadequacy to meet the standards set by my illustrious predecessors in office. What I did not fully appreciate at that time was that I had been made the titular head of an altogether surprising organization. I would have been spared my solicitude could I have realized how soon I was to be absorbed by its smooth functioning machinery. In every department I was to find friendliness and efficiency.

As one contemplates the history of The New York Academy of Medicine one can, without stretching one's imagination too much, divide the story into four fairly distinct chapters. Chapter One would include that period dating from the adoption of the original constitution on January 6th, 1847 and ending with the occupancy of its first home in 1875. Chapter Two would comprise the years between 1875 and 1890, the latter date representing the opening of the first building actually constructed to meet its growing needs. Chapter Three is most important because it tells the story of the early days of what might be called the modern phase of our development. It begins in 1890 and continues for over thirty years. It is my conviction that one of the most far-reaching undertakings in the Academy's long history occurred in this period,

* Delivered January 7, 1943 at the Annual Meeting of The New York Academy of Medicine.

namely, the formation of the Committee on Public Health Relations in 1911. The service which this committee has rendered is told in a small volume entitled "Thirty Years of Community Service 1911-1941." Of the original membership but one is still living, my immediate predecessor in office, a former chairman of the committee and its present presiding officer, Dr. James Alexander Miller.

The Fourth Chapter began in 1924 with the conclusion of an agreement with the Rockefeller Foundation for an extension of the educational activities of the Academy of Medicine. The essential features of the plan were:

a. A suitable new building
b. Development of the library
c. An efficient information service for visiting physicians—American and foreign
d. A similar information service for the medical profession of Greater New York and vicinity
e. A development of medical extension activities at the Academy building
f. Coöperation with hospitals to provide clinics, etc. throughout the city for the profession
g. Promotion of popular education with regard to preventive medicine
h. The appointment of a special educational committee of the Academy
i. A director of educational work on a full-time basis

The Carnegie Corporation very generously provided for the building.

To these two foundations the Academy and the community owe a debt which is difficult to express in words. It is a satisfaction to know however that they feel that we have justified the faith they had in us.

I shall not attempt to develop the story of the current chapter in Academy history. It is sufficient to state that three of our four standing committees had their origin during this era. The Committee on Medical Education in 1927, the Committee on Sections in 1929 and the Committee on Medical Information in 1939.

This fourth chapter is still being written but I am inclined to believe that a new or fifth chapter is about to be initiated.

Just as chapter four was in part a product of World War I, so in

my opinion chapter five will be written as a direct result of the present World War. Social and economic trends will affect every phase of life. Medicine will face a vastly changed world and medicine must take the lead in determining just what its position is to be in the new order or else the initiative will surely pass to those less qualified to act. The Academy of Medicine is essentially a teaching institution with public health forming the basis for its chief interest in this direction.

It is a matter for regret that a feeling seems to exist in some quarters that the Academy of Medicine does not coöperate as it might with the Medical Society of the County of New York. This is due I think to a failure to recognize the fact that while the two organizations have a great deal in common, nevertheless their aims are in some respects quite different.

The Academy of Medicine maintains a public library free to all during certain hours of the day. Around this feature there have been slowly developing for nearly a century certain educational features which have made The New York Academy of Medicine a distinctive and unique organization. Every standing committee of the Academy is interested in some phase of postgraduate medical education.

It is also well to remember that nearly every Fellow of the Academy of Medicine is also a member of the Medical Society of the County of New York. We therefore appreciate and have full understanding of the social and economic problems with which medicine is surrounded. We as individuals are a part of organized medicine.

This Academy of ours is no "roost where emeritus professors and effete men of letters once cocks of the walk could sit in quiet rows while the fighting was going on beneath them," to quote again a portion of a letter written by Oliver Wendell Holmes on the occasion of the opening of the new Academy Building at 17 West 43rd Street. On the contrary you will find us an active body of men and women giving freely of ourselves for the benefit of the public welfare in matters bearing the slightest relationship to its health. After four years as your president surely I am in a position to state that The New York Academy of Medicine wishes to give full coöperation to the Medical Society of the County of New York wherever it may be found that our objectives run parallel.

Another criticism which has been brought to my attention recently concerns the method employed in selecting candidates for Fellowship in

the Academy. The Committee on Admission is nominated by a group of Fellows appointed by the President for that purpose. Four men are elected each year to serve a term of three years. Thus this committee consists of twelve Fellows duly elected by the Academy at annual elections. During my tenure of office I have attended most of the monthly meetings of the Admission Committee and I have been greatly impressed by the serious consideration given to each candidate for Fellowship. As Academy prestige has increased in the last twenty-five years the qualifications for Fellowship have also undergone change. We are as interested in keeping unqualified candidates out of Academy Fellowship as we are in electing candidates with satisfactory background.

The Committee on Admission represents a cross section of the highest ideals in the medical profession and I can assure you I am as satisfied with the rules under which this committee functions as I am with the way these rules are put into effect.

It would not be possible for me with the time at my disposal to relate more than a small fraction of the activities of the Academy during the past year.

The Library Committee continues to maintain the library of the Academy in a high state of efficiency in spite of the war. Its records have been protected by microfilming. Its rare books are not only covered by war damage insurance but steps have been taken to give physical protection to these irreplacable treasures. We continue to serve more readers than any other medical library on this hemisphere and possibly more than any other medical library in the world at the present time. We are greatly in need of more stack room and work space but these cannot be furnished until we have finished with the Axis powers.

The Committee on Public Health Relations is a civic institution in its own right. During the past year it has released for publication a large and comprehensive volume entitled "Preventive Medicine in Modern Practice."

I would invite you to read the small monograph entitled "Thirty Years of Community Service 1911-1941," if you would familiarize yourselves with the variety and importance of the services rendered in the past by this committee.

The Committee on Medical Education conducted for the Academy a highly successful Graduate Fortnight in spite of the war and an anticipated reduced attendance and I quote from the report of the Director,

"At no time during the fifteen years in which these meetings have been held has the Fortnight Committee more richly deserved the appreciation of the Academy Fellowship and the medical profession at large." The exhibit this year was especially rich in material and reflected great credit on the committee under whose supervision it was assembled. Few of us appreciate how much we owe the members of this Committee on Education for the time and thought expended by them in giving us such lectures as we are privileged to hear on Wednesday and Friday afternoons and for the splendid programs provided for the Stated Meetings of the Academy.

An arrangement has been made by this committee which will increase the circulation of the Spanish and Portuguese language medical journal, America Clinica, so that it will reach fifty-two thousand Latin-American practitioners.

The Committee on Medical Information has coöperated with the Town Hall in repeating six lectures on contemporary medicine. At the request of the Department of Education and Superintendents of Schools thirty lectures for teachers were arranged on the subject "Child Health in War Time Conditions." The third annual one day Health Conference was conducted by the committee and the transactions of the second annual conference were published. This committee also published a "Study of Radio Broadcasting" made under a fellowship obtained from the Rockefeller Foundation, Division of Humanities. The Laity Lectures continue to fill a most important place in our scheme of things. The seventh volume is about to be published. I commend it to you without reservation.

The Committee on Sections has been completely reorganized in the past two years under the guidance of a special committee appointed by direction of the Council.

The war has taken over fifteen per cent of our Fellows into the various services of the country. This has meant not only serious loss in personnel from the staff and the standing committees but also a considerable loss in income from dues. However, the slack has been taken up by those who for one reason or another have been obliged to remain at home.

The special committee composed of laymen and Fellows who have worked so faithfully to give the public an understanding of what your Academy is doing in contributing to its health and welfare deserves a special word of appreciation. Their effort will continue to bear fruit.

Let me go back sixty-five years to quote from the presidential address of Dr. Samuel S. Purple on taking the chair as president for a second term on January 18th, 1877. He had the faith that we of our generation are justifying.

> ". . . Does the medical world belong only to the generation which inhabits it? Is it not rather an entailed estate, the income of which the present possessors have the right to enjoy, but not the right to squander or scatter? Are they not in honor bound to preserve the estate intact, institute and develop such permanent improvements as will tend to meet the wants of the generations which will follow? Such are the dictates of a general philanthropy, which emanate from a proper love of mankind, and have the approval of that sound judgment which is strengthened by observation and matured by age. Let not, then, those who have labored for years past to build up here an institution that shall advance the best interests of medicine, and at the same time be as free as possible from the foibles of the profession, become weary in heart; the design is being steadily carried into effect; the end can be surely reached—the object will, by persevering effort, be accomplished."

My task as your President is done. I shall never cease to be grateful to the men I have worked with. I have been carried along on the wave of enthusiasm which pervades our institution, an inheritance from former generations of Fellows. I shall never lose my devotion to the interests of the Academy. I am not going to single out particular individuals on whom to bestow words of praise. It would be a long list and consume much too much time.

I now take great pleasure in presenting to you your new president. He needs no introduction for he has served you long and well as Fellow, Committeeman, Vice President and Chairman of the Board of Trustees. I know of no man better qualified than he both by character and experience to guide you, your President, Dr. Arthur Chace.

THE ACADEMY MEETS THE CHALLENGE OF THE FUTURE*

ARTHUR FREEBORN CHACE

President of The New York Academy of Medicine

Inaugural Address

WE are inspired by the realization that a physician should have considered it an honor to give all his time for two terms to this noble institution, after having devoted years to the advancement of its work. This, Malcolm Goodridge has done and tonight he ends four years as President of the Academy.

Fortunately for us, it is not an ending but a continuation, for we feel sure that Dr. Goodridge will always share with us our responsibilities and give to us the accumulated wisdom gained during a lifelong service to the Academy, a service which so eminently fitted him for the years as our President.

During this time he has devoted all of his strength and energies to the solving of the many difficult problems which have confronted us. His patient attention to detail, his never-failing faith in the ability of the Academy to meet its ever-increasing burdens, his unselfish devotion and wise leadership will always be an inspiration to those who follow him.

One cannot contemplate the future either as it may affect the life of an individual or that of an institution without being acutely aware of the great emergency which confronts us individually and collectively. From whatever vantage point we survey the effects of the great storm now sweeping the world we cannot escape the whisperings of anxiety nor deny the imminence of danger. Civilization, even all that the Academy holds most valuable, is threatened with well-organized destruction by men who have wilfully and purposely reverted to barbarism.

* Delivered January 7, 1943 at the Annual Meeting of The New York Academy of Medicine.

In Paris, Louvain, Warsaw and in London, libraries have been pillaged or lie in ruins. Books, the very lighthouses of our profession, by which we can steer our course true into the future, have been piled in heaps and burned. In Europe the free interchange of ideas and discoveries, through which science has grown great, is no longer possible.

We cannot hope to penetrate even lightly the mist of the future without casting upon it that illumination which can be gained through the contemplation of the past and the present in historical perspective. The New York Academy of Medicine is now in its 96th year. During these years it has confronted and surmounted many crises. It has shared in the destinies of our people and our country and has, through its constructive work, helped to shape the issues of the years.

The Academy not only survived the internecine strife of the Civil War which broke out shortly after its creation; the trials of the long Reconstruction period and the ordeals of the last World War, but has emerged from these tests richer by the experience and stronger by the trials. This was possible because of the far-seeing wisdom of its leaders and the faith of its fellowship. Both recognized that virile life ever necessitates, as Herbert Spencer so aptly phrased it, the adjustment of inner relations to outer circumstances. The Academy has never wavered in its devotion to its objectives which were the reasons for its creation; to foster the science of medicine and to maintain the highest ideals in its practice. Wisely, however, the ways in which the Academy has pursued these objectives have changed with time. They have grown in scope and in complexity even as did our science and our social and economic existence.

The Academy has undergone a series of revolutionary progressions. In the earliest years of its existence, it was chiefly devoted to the collection of a library where men might read and learn. Then, as the need for discussion in common impressed itself upon the fellowship, there developed a strong drive for the procurement of a permanent home for the Academy. Still later, and that brings us down to the beginning of the present century, as our community grew in complexity and as our science developed infinitely, it became evident that precious as was our library and useful our meeting halls, they could not serve to fulfill the growing obligations of medicine to society. In that way, thirty-one years ago, there came into being the Public Health Relations Committee, eighteen years ago the Committee on Medical Education, and more

recently the Committee on Medical Information.

These developments and achievements so briefly summarized, were not realized without great effort nor without contest even in our own fellowship; "the adjustment of their inner relations to outer circumstances" is never an easy accomplishment. Yet that they were achieved is witnessed in our history and with what profound benefit to the medical profession and to the public, is attested by all. The Academy will continue to collect, preserve, distribute, and advance all that is best in medical skill. For, in the words of Hippocrates, familiar to you all: "*The physician must know what his predecessors have known, if he does not wish to deceive both himself and others.*"

What is there, then, in the knowledge of our predecessors which will help the Academy to meet not only the challenge of the present but also the still greater challenge of the future? More than two thousand years ago Heraclitus laid down what he believed to be one of the fundamental laws of the universe. "*There is nothing permanent*," he said, "*except change.*" Certainly science has found little to disprove that assertion and much to confirm it.

Some medical men, influenced by Darwin, have tended to believe that, through the elimination of the unfit, the forces of Nature move continuously upward and onward. They see now what they had almost forgotten, that during certain periods the body politic, like the body human, may stagnate—even retrogress. The undesirable and destructive elements are in the ascendant and although change never for a moment ceases, it is not *ipso facto* to be assumed that the change is for the better. "*All that is human*," said Gibbon, "*must retrograde if it do not advance.*"

This then, I humbly submit, is the beginning of wisdom for the Academy in these perilous times. But despite the fact that half the world is in flames, *we* do not have to retrogress. It is our sacred duty, our inescapable destiny, to advance, eyes open, into the future, there to meet whatever challenge life shall hold.

Medicine being of the web and woof of our social life shares in all the changes and also in the anxiety with which all mankind faces the future. Forthwith, however, some of this anxiety can be dissipated in the realization that whatever else betides the world, as long as there are men on this earth, there will be illness among them and as long as men are ill they will be in need of the best care that medicine can afford them and will want that care administered to them in the best tradi-

tions in which the Art is to be practiced.

In this we have much to fortify us. In the last seventy years medicine has achieved more scientifically than it had in the rest of recorded time. More freely than ever before "the never-idle workshop of Nature" has been yielding up its secrets to man. To the successful use of the sulfonamides has been added the discovery of the most dynamic vitamin, Biotin. In the past few years the hypothesis of the atomic theory has been strengthened by the splitting of the atom. In the case of uranium the amount of dynamic energy thus released has actually been measured. We are on the threshold of solving many riddles, among which it seems safe to place such virus diseases as infantile paralysis, the common cold and the newer form of virus pneumonia.

But here we are confronted by what appears to be another universal law that behind each advance of science lurks some new and concomitant problem. No sooner does the profession feel reasonably confident that the cure of pneumonia has been found than a new, virus-type form appears. It does not respond to the new treatment. Medicine must then immediately institute new research, hopefully employing gramacidin and penicillin.

The United States is fortunate today in that many of the greatest scientists of the modern world have gathered here under the standard of liberty and will join our scientists in this basic research.

Life expectancy at birth has during the past twenty years been extended by at least ten years through the work of modern science. The problem thereby created is the treatment of the largely increased illnesses associated with old age. The development of the specialty of Geriatrics is a pressing problem wherein the Academy must forge ahead in its educational program, fitting physicians to meet this demand, else we would not uphold Browning in his plea:

"Grow old along with me—
The best is yet to be!"

Physical science, however, has advanced much more rapidly than have our social sciences, and to this lag may be charged many of the problems that are facing us today. Our biological, chemical and physical discoveries have far outrun our social and economic adjustments and the war demands have completely thrown society out of gear. In no other branch of science as in medicine have its discoveries been so quickly, and at so little cost, put at the disposal of the public; but still

it is not enough. In this lies the Academy's opportunity and obligation to educate the medical profession for their proper use and the laity for proper coöperation.

The approach of the Academy to medicine has always been—and must continue to be—through the individual patient. This is, in many respects, the more difficult way. But for society as a whole it is, infinitely, the surer way.

It is also, I believe, the future way. Two recent portents will confirm us here: Lord Nuffield recently endowed for a period of ten years, the Institute of Social Medicine at Oxford—an interesting and significant event which has received too scant public notice. Let me quote the exact words of the announcement of the Institute's objectives:

"*To investigate the influence of social genetic, environmental and domestic factors on the incidence of human disease and disability.*

"*To seek and promote measures, other than those usually employed in the practice of remedial medicine, for the protection of the individual and of the community against such forces as interfere with the full development and maintenance of man's mental and physical capacity.*"

In the same spirit, but with a somewhat different objective, should be mentioned the creation of the Nutrition Foundation by a group of food and other, closely related manufacturers. This foundation has assigned a fund of a million and a half dollars to support a five-year program in the application of the science of nutrition.

We have, apparently, some companionship in going out to meet this challenge. On the one hand we have a philanthropic University foundation; on the other a purely industrial foundation, both supported by large sums of money and both devoted to the application of the medical sciences to the improvement of the well-being of man.

We have an ally more precious still. Never before has the public been more interested in the scientific and the sociological aspects of medicine. No "social architect" can meet this hunger because it is not a hunger for more social theories. It is rather a hunger for established facts, truthfully correlated and interpreted in the impartial light of science. Society's ills, as the public instinctively feels, are not going to be cured by the anodyne of wishful thinking. They are beginning to see that the first step is the same as in the treatment of disease in an individual patient. We must clearly define the disorder, its symptoms and its probable causes. Then and then only may we recommend such

remedies, such changes in environment, as will remove the morbid manifestations by eliminating their causes.

It would be presumptuous for any man to pretend that, standing in the midst of the storm, he can diagnose with full accuracy the nature of society's present global difficulties, their causes and their cure. Yet the medical profession, standing on the solid base of known fact and trained, as it is, to maintain calm in the face of any disaster, will be able assuredly to make a worthwhile contribution.

The humanitarians of the last century, the Chadwicks and the Shattucks, could do no more than protest angrily against the degrading circumstances in which so many men labored and lived. But not until modern medicine revealed the microbic, the diet deficiency and other causes of disease, could their socially-righteous indignation be implemented and rendered effective. It is not enough to *feel;* it is essential also to *know.*

As long ago as the Seventeenth Century, Shakespeare could have King Lear say,

> *"Poor naked wretches, wheresoe'er you are,*
> *That bide the pelting of this piteous storm,*
> *How shall your houseless heads and unfed sides,*
> *Your loop'd and windowed raggedness, defend you*
> *From seasons such as these? . . ."*

He could ask the question but there was not, at the time, a man, nor any group of men, anywhere in the world who could give answer. They simply did not know.

Today, at least, medicine can offer a clear and valid knowledge on many fronts—fronts which it is our duty to expand. To every segment of man's life, whether it be at home or in the work place, on the battlefield or in the metropolis, medicine has been and is making fundamental contributions in knowledge and in guidance. We have made vast advances on the physical front and some, but not enough, on the psychological front.

I would not add even so much as one to the thousands of definitions of Man. But on the fact that Man is above all *a thinking animal,* all of us would be in agreement. Part, at least, of the challenge of the future to the Academy, therefore, is that it must think more clearly and deal more thoughtfully not only with man the corporeal being, but also with man the cogitating creature.

Yet here we must be aware of a pitfall which has engulfed so many of our contemporaries. We must not be tempted into drafting a blueprint for salvation; one of those schemes which are compounded out of wishful thinking and the naive belief that given but enough enthusiasm we could—

> "..... conspire
> To grasp this sorry Scheme of Things entire,
> ..... and then
> Re-mould it nearer to the Heart's Desire!"

We need rather to follow the same pattern that serves us when we deal with the ills of man. And in all this we must cleave to that wisest of all therapeutic precautions "*above all—to do no harm.*"

It must be patent to everyone that much of our difficulty can be charged to the enormous disparity that exists between our scientific and technological advances and our social and economic evolution. In the light of what *we know* we are woefully backward in what *we do.* We must then catch up with these advances realizing that the crux of the problem is *how* this catching-up is to be effected.

To this problem medicine with its vast encompassments can and must make a solid contribution; for medicine can contribute its knowledge to the thinking and planning of the social architects who are ambitious to build the society of tomorrow. Today there is a vast and potent body of medical knowledge that can serve to make the world a better place to live in. For medicine, notably during the present century, has extended its sway far beyond the mere diagnosis and treatment of illness. It has gained precious knowledge about the normal operations of the human body, and, no less significantly, the normal, as well as the abnormal operations of the human mind and psyche. Today, better than at any time in the history of mankind, we know how to promote and to conserve human well-being physically, mentally and emotionally.

We call in witness of this the enormous strides that have been made in the specialties of nutrition and psychiatry. In place of those truly fantastic speculations about man and his social relations, initiated by Rousseau and since elaborated by many different schools of thought, medicine today offers a much clearer and infinitely more valid body of knowledge on the psychological mainsprings of human behavior. It is no accident that in the world strife of today this knowledge has been enlisted in the propaganda battle between the warring nations.

It may seem that we have wandered far from the title of our paper, *The Academy Meets the Challenge of the Future*, but in substance we have not. For in all those "affairs of today and tomorrow" which have been described, those which are in substance "the challenge of the future," the Academy has an important role to play. For the "social architects" will not entirely and on their own initiative come begging for the guidance and instruction that medicine affords.

It is, therefore, necessary that medicine should exercise its influence actively, not with the aggression of a "pressure group" but rather with that positiveness which is warranted by its knowledge. This is no new function, nor yet a novel role for the Academy. For more than three decades the Academy has through its Committee on Public Health Relations, through its Committee on Medical Education and more lately through its Committee on Medical Information, played an ever-increasing role in dealing with problems of public policy.

We are resolved to continue and to intensify these activities. More specifically, though without elaborating the details too largely, we at the Academy propose to institute in the immediate future, through our standing committees and through such special committees as will be required, careful and thorough-going studies of the trends and the indicated future developments in medicine and in communal and public health. We propose to scrutinize carefully the proposals and plans for the post-war world that are formulated by responsible and authoritative agencies. We intend to do this so that we may be fully informed and in a position to contribute effectively to such services as we may be ultimately called on to render. It is likewise our intention to devote thought and consideration to the more immediate problems that confront us in the medical world, to those that stem from the rapid changes that are taking place in the undergraduate education of the physician, from the reorganization and the administrative changes of the voluntary hospitals that are dictated by the curtailment of financial support, from the withdrawal of so many physicians from civil practice to military service, from the relocations of populations, and we must also devote our attention to those various health problems that issue from food and fuel rationing, from the intensification of our industrial efforts, and from the increased participation of women in war work.

Now once again, with pressing urgency, demands are made upon us. We are meeting—we must continue to meet—these demands. Civilian

Defense Units must be kept up to date. Catastrophe Teams must be kept continually on the alert. Plans must be perfected for the emotional and physical rehabilitation of men returning from the war. The Refresher Courses must be given with increasing intensity. Yes, many a specialist must now reverse his steps and, with eagerness, set out to become the improved counterpart of the Horse and Buggy Doctor.

If only one-half of the former number of active doctors remains in New York to look after the entire problem of public and private health, then men over forty must renew their youth in vastly-increased service to the community.

The Academy must add to its responsibilities without relinquishing a single one of its activities in the State or Nation.

These, then, are the challenges of the future. These, we must go forth to meet. *The fellows of this great institution must be not only defenders of the noble traditions which they have inherited, they must also strike out fearlessly into the unknown.* We must purpose to be more than guardians. We must resolve–

"To follow knowledge like a sinking star.
Beyond the utmost bound of human thought."

I would not have you believe that ours is an easy task. Encompassed about on all sides by destruction, by change and by manifold anxieties we must nevertheless continue–

"To strive, to seek, to find, and not to yield."

We must so live and so achieve that, fifty years hence, members of this august body, looking back, will say, "Those were great years in the Academy's history. In troublous times, they held the torch aloft."

When others falter, or lose the way, the Academy must–the Academy will–point the way and define the path which the medical profession should follow. Physicians, true to their calling, will be inspired by the difficulties and burdens that await them and with one accord give unsparingly of themselves. For, as Hippocrates truly said, "*Wherever the Art of Medicine is loved, there also is love of humanity.*"

We live in a changing world. We live in a world at war. We must realize now, more than ever before, that–

And we must take the current when it serves.
"On such a full sea are we now afloat.
Or lose our venture."

A RESUMÉ OF THE PRINCIPAL DIAGNOSTIC FEATURES OF SUBDURAL HEMATOMA*

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CEREBRAL subdural hematoma is a lesion essentially of traumatic origin, occurring with increasing frequency in the mechanized era in which we live, producing symptoms at variable periods following injury and should be entertained as a possible complication of every craniocerebral trauma however trivial the accident may have seemed. As holds true for the advancements of our knowledge regarding any particular clinical entity, a few outstanding contributions concerning subdural hematoma have been responsible for the progress that has been made in the treatment of patients with this pathological process. It was in 1857 that Virchow[1] reviewed the previously proposed concepts relative to encapsulated hemorrhages in the subdural space and set forth his own thoughts regarding the pathogenesis of this lesion. This article appears to have influenced medical thinking for the next fifty years. In any event during this period it was quite generally held that blood clots disclosed in the cerebral subdural space were chronic in character and existed for the most part in idiots, the senile and the insane. In 1905 Bowen[2] presented a study of 72 cases of subdural hematoma reported in the literature between 1870 and 1900 and adduced evidence indicating that there was a causal relationship between trauma and this lesion. This excellent presentation of the subject remained relatively obscure and it was not until twenty years later that Putnam and Cushing[3] in 1925 focused the attention of the present generation of American surgeons on a pathological state that could be cured by appropriate surgical measures. Beginning in 1934 Munro[4] contributed a series of articles dealing with the pathogenesis, the diagnosis and the surgical treatment of the varied subdural collections that complicate craniocerebral injuries. His work has been important in that many problems relative to pathogenesis have been studied in considerable detail. Contributions by

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Frazier,[5] Peet and Kahn,[6] Jelsma,[7] Ingraham and Heyl,[8] Kaplan,[9] Kunkel and Dandy,[10] Leary[11] and others have advanced our knowledge concerning this lesion.

In striking contrast to the ideas held during the nineteenth century it is now well substantiated that trauma to the head is by far the most frequent etiological factor in the production of subdural hematoma, however, these lesions may be the result of spontaneous hemorrhage. I have observed examples of bleeding into the subdural space from cortical vessels implicated by a metastatic tumor of the brain, from ruptured cerebral aneurysms, from an arteriovenous malformation on the surface of the brain and from the region of subcortical abscesses. In most instances a moderately severe traumatic cerebral insult has been sustained with resultant contusion-laceration of a temporal and/or under-surface of a frontal lobe. Bleeding from lacerated vessels in one or the other of these damaged areas collects in the subdural space. Or a vein crossing from the cerebral cortex to the dura or leading into one of the dural sinuses may be torn with the production of a large blood clot. In some it is impossible even at autopsy to identify the source of the hemorrhage.

Recently established collections in the subdural space may consist of solid blood clots, blood admixed with cerebrospinal fluid or xanthochromic fluid in considerable quantity, the so-called subdural hydroma. Slowly a membrane is formed about the collection, that portion of the capsule on the dural side becoming relatively thick and quite vascular, whereas the membrane adjacent to the arachnoid is thin and devoid of prominent blood capillaries. The accumulated evidence indicates that coagulated blood usually undergoes liquefaction and, as pointed out by Gardner,[12] adjacent fluid (cerebrospinal) is drawn into the subdural collection producing an increase in the size of the intracranial mass. The clinical course of many patients suggests that the subdural mass may enlarge several days, weeks or sometimes months after the accident or, at any rate, symptoms put in their appearance at these late dates. Occasionally the clot is invaded by fibroblastic elements with a resultant organized mass. Yet patients in whom this has occurred may manifest the same delayed symptomatology as observed in those with encapsulated fluid collections. It seems unlikely that an organized blood clot could expand within a few days after being relatively quiescent for many weeks. Alterations in cerebral circulation in the vicinity of either an organized blood clot or the chronically encapsulated fluid collections

are more likely the explanation for the symptoms. In support of this concept specimens obtained at autopsy have shown obvious edema of the brain adjacent to an encapsulated fluid hematoma. This local edema could be logically assigned as the cause of the clinical signs which had been demonstrable. The organized blood clots and the more commonly encountered encapsulated liquid subdural collections are the lesions that have attracted the attention of neurological surgeons and concerning which excellent discussions have been recorded. These collections are not infrequently disclosed at operation, under the mistaken diagnosis of brain tumor. They are found in patients who have survived a mild to moderately severe head injury which in many instances has long since been forgotten. They have been termed chronic subdural hematoma, a designation entirely consistent with the duration of the lesion, however, the term should not be used to imply that this syndrome has a separate and distinct pathogenesis from the so-called acute subdural hematoma. The chronic state is merely a latent variant of the acute. Moreover the patients with chronic collections present symptoms and signs that are readily interpreted as representing a space-taking mass in the head and if not localizable by clinical findings, their position can be accurately determined by Dandy's method of ventriculography. The mortality from operative removal of these chronic lesions should be almost nil, therefore, further discussion concerning this entity need not detain us here.

The acute form and the subacute variety, if terms signifying duration of the lesion are permissible, are the commonest types of subdural hematoma. Some patients with this lesion die within a few hours following injury, some survive for several days in a state of continuous stupor, others rouse from stupor after three or four days only to lapse into coma in the next eight or ten days, and still others less severely injured become oriented two or three days after injury, remain so for two or three weeks albeit complaining of headache, then backslide into a confused state. While it is recognized that the subdural collections associated with these syndromes are often a single complication of a general brain insult and surgical removal may not favorably influence the eventual outcome in some that are severely injured, nevertheless, prompt recognition of the probability of such a lesion, the employment of appropriate diagnostic measures and the execution of judicious surgery will save many who otherwise would succumb.

During the past eight years there were admitted to the Kings County and Brooklyn hospitals 18,272 patients with varied types of craniocerebral injuries. Among these there were 289 instances of subdural hematoma. A study of 143 of these (five year period) has been previously reported.[18] Experiences with the entire group of 289 cases has led to the conclusion that an accurate diagnosis is seldom possible from the clinical features alone. In by far the majority of instances there were associated intrinsic brain lesions productive of abnormal physical signs which served to confuse the diagnostic issues. There are, however, a few clinical observations that are significant and are indispensable aids in arriving at a working diagnosis. These are: (1) the external evidence of trauma to the head and an estimation of the area receiving the maximum blow, (2) the state of consciousness, (3) the condition of the pupils, especially inequality, (4) weakness of an extremity or extremities of one side and (5) the status of the superficial and deep reflexes. Aside from the verification that a blow to the head has been sustained, the observations relative to the conscious state are most important. A short period of drowsiness or stupor, the consequence of the initial insult, followed by enduring orientation is seldom associated with subdural hematoma. However, a period of a day or two of confusion or drowsiness followed by several days to weeks of relative lucidity, during which the patient's intellect is mildly blunted, and then the supervention of progressive drowsiness to stupor, is highly suggestive of a subdural collection. Profound stupor following a craniocerebral injury, with or without physical signs indicative of a focal lesion of the brain, that continues longer than 48 to 60 hours also indicates that all is not well and the patient is in need of further diagnostic and therapeutic measures.

The condition of the pupils, especially inequality, has not been found to be as helpful a guide as reported by some. The pupils were recorded as unequal in 46 per cent, or 132 cases, of the series. In 29 per cent of these 132 cases, the larger pupil was on the opposite side to the subdural collection. In only 11 per cent was there found an unequivocal widely dilated pupil on the same side as the lesion. It is therefore evident that one may be misguided by attaching undue importance to the presence of unequal pupils.

Weakness of an extremity or extremities of one side was observed in 166 of the series. The hemiparesis was present on the same side as

the subdural hematoma in 64 of these. In other words, 22 per cent of the entire series of 289 cases had weakness of the extremities on the same side as the subdural hematoma. The presence of motor weakness of the extremities of one side following a craniocerebral injury does not necessarily indicate that the cerebral hemisphere of the side opposite the weakness is being compressed or otherwise functionally altered by a blood clot. If a hemiparesis be demonstrable, however, the possibility of an intracranial hematoma should be entertained and the diagnosis of such a lesion be established or excluded by small multiple cranial openings and/or ventriculography.

The reflexes, both superficial and tendon, are frequently found to be abolished shortly after a moderate or severe craniocerebral trauma. Babinski's sign may be present bilaterally. After recovery from the immediate effects of the insult the tendon reflexes may be obtained, although such bizarre reflex findings are frequently present that interpretation is difficult. In truth, observations relative to semi-purposeful or random movements of an extremity or extremities are more helpful in estimating disturbances in neural mechanisms than are refined tests.

Records of the pulse rate, respiratory rate, blood pressure and the cerebrospinal fluid pressure and its characteristics should be kept, but these singly or in combination are not to be considered as reliable indices for therapy. They are only a part of the entire clinical picture and are to be evaluated accordingly.

From this abbreviated account it would seem that time honored clinical findings derived from physical examination are wholly unreliable. In many instances this may be true and the evidence obtained from ventriculographic examination supersedes all the other observations. Although this form of examination is many times an indispensable aid in establishing or excluding the presence of a subdural hematoma or other intracranial blood clots, one does not inject air into the cerebral ventricles of every patient who is in an unconscious state. It is still important to arrive at a reasonable clinical diagnosis before subjecting the seriously injured to an operative procedure however minimal. Familiarity with the clinicopathological syndromes that are commonly encountered is therefore essential for differential diagnosis.

As previously alluded to, multiplicity of cerebral lesions in the same patient is one of the greatest obstacles in identifying the presence of a sizable blood clot that should be surgically removed. It is often impos-

sible to assign with certainty the etiological factors to obviously abnormal neurological states. The clinical features of the common syndromes may be elaborated upon to advantage. There should be no question regarding the therapeutic course to pursue in any patient who has sustained a cerebral insult with or without immediate loss of consciousness followed by a period of relative lucidity and this superseded by drowsiness, stupor and deepening coma. 'Tis true that exceptions are encountered; however, under these conditions the advent of drowsiness with or without focal signs is the signal for prompt surgical intervention. This is the story of slow arterial bleeding in the intracranial cavity. The blood clot may be located epidurally, subdurally or within the brain itself. The abnormal physical findings are often insufficient to localize the lesion with precision, therefore ventriculography is essential before any operative procedure is carried out. It is unwise and unsafe to attempt the removal of hematomas, be they epidural, subdural or intracerebral, through a traditional subtemporal decompression opening. If the subdural collection is not entirely liquid and cannot be evacuated through two small cranial openings then it is preferable to utilize a small bone flap made over the site of the lesion as demonstrated by the air studies.

There is a second clinical syndrome that calls for prompt and precise diagnosis followed by appropriate surgery according to indications. The following is an illustrative story: A patient who has sustained a moderately severe craniocerebral injury is admitted to the hospital shortly after accident. He is drowsy, disoriented and non-coöperative. The pupils are equal, the extremities are moved about equally well and no abnormalities in reflexes are demonstrable. Slowly over a period of eight to twelve hours stupor supervenes and one pupil is found to be slightly larger than its fellow. Pinching the pectoral muscle border or application of other painful stimuli evokes acceleration and increased depth of respirations and transitory skeletal muscle hypertonicity with all the extremities in a fully extended and rigid posture. Shortly, this rigid state, or so-called decerebrate rigidity, becomes almost continuous, the breathing is labored and forceful, the jaw set and the facial muscles contracted, simulating somewhat the sardonic grin of tetanus. The temperature rises and the pulse becomes accelerated. The skin is flushed, at first dry and hot, later the patient is bathed in his own sweat. Such a course of clinical events has been observed in association with a wide

variety of lesions: epidural, subdural, intracerebral or intraventricular hemorrhage, diffuse small hemorrhages throughout both cerebral hemispheres often associated with contusion/laceration of one or both frontotemporal regions, and/or hemorrhages in the upper part of the hind brain. The pertinent point relative to the present discussion is that a large subdural hematoma may be the cause of this clinical syndrome. While it is known that the decerebrate or decorticate attitude, regardless of the causative factor, is a bad prognostic sign, nevertheless, we have had a number of recoveries following the removal of large blood clots in patients manifesting these clinical features. It has been observed that inequality of pupils and the decerebrate attitude are more commonly associated with large supratentorial hematomas than are equally constricted pupils and the decerebrate attitude which are not infrequently found in association with hemorrhage within the upper hind brain. In all events the appearance of decerebrate rigidity in any patient who has sustained a head injury calls for ventriculographic examination unless a surface hematoma be disclosed in the making of bilateral openings for purposes of this examination. Further surgery is carried out in accordance with the information derived by inspection through the cranial burr openings and/or the aerographic study.

The third group of patients with craniocerebral trauma complicated by subdural collections comprise those that remain confused, drowsy or stuporous from the time of injury until the hematoma is surgically removed and/or death ensues. The obvious lesions of the brain disclosed in those that come to autopsy are single or multiple surface contusions or lacerations with regional edema. As previously stated a surface vessel, often a vein, has been torn at the site of the cerebral contusion and bleeding has occurred into the subdural space. Approximately two-thirds of all subdural hematomas are the result of this type of lesion. The clinical course of patients so injured is quite variable, dependent no doubt on the pathophysiological alterations within the brain itself. At least the marked changes in temperature, pulse rate, respiration, etc. suggest primary dysfunctions of the vegetative system. On the somatic side, hemiparesis or hemiplegia are not uncommon. Here again the exact role played by the subdural collection in the production of motor weakness of the extremities is difficult of evaluation. Often a lesion within the brain appears to be the more important. Be this as it may, patients severely injured frequently die within twelve to thirty-six hours follow-

CHART I

Number of subdural hematomas operated 227
Number of subdural hematomas non-operated but verified at autopsy.. 62
Total number of subdural hematomas (operated and non-operated).... 289

Time elapsed between Injury and Operation	*Total*	*Recovered*	*Died*	*Mortality*
2 to 24 hours	51	9	42	82%
1 to 7 days	65	34	31	48%
7 to 14 days	48	36	12	25%
14 to 21 days	33	26	7	21%
21 to 28 days	11	8	3*	27%
Over 28 days	19	17	2	11%
Totals	227	130	97	42%

* One of these had a subdural collection associated with multiple metastatic foci of infection in the brain.

ing the accident. Surgical removal of a subdural hematoma carried out during this critical period seldom alters the situation in a favorable manner. In fact we have come to believe that the removal of hematomas during the first twenty-four hours after injury is seldom justifiable. Chart I clearly illustrates the reasons for this position.

This chart shows the progressive decrease in the mortality rate following operation in those that were successfully carried through the critical period after injury. The sixty-two patients who died without operation represent those admitted to the hospital in a moribund state. Many of the patients from whom subdural hematomas were removed during the 48 hour period following injury presented evidence of severe brain damage. Among these, operation was a "last-resort" affair in many instances.

If the employment of supportive measures will not tide the patient over this critical period, then little should be expected from surgical intervention. Those less seriously injured usually weather the storm. However, if after a few days of progressive improvement there ensues drowsiness or untoward alterations in vital signs, the presence of a subdural hematoma should be established or excluded by making multiple small cranial openings and/or ventriculography. If a hematoma is disclosed by these examinations it should be promptly removed regardless of its anatomical position.

Summary

It has been well established that in the majority of instances trauma is the cause of cerebral subdural hematoma. The varied alterations that take place in the size and consistency of many of these blood clots is still not well understood. Furthermore, the role played by cerebral edema adjacent to a chronic encapsulated hematoma in the production of symptoms and signs is also in need of further consideration. The clinical manifestations attending craniocerebral injuries complicated by subdural hematomas are difficult of interpretation. Multiplicity of lesions within the intracranial cavity is the rule rather than the exception. A few clinical syndromes are encountered, however, that suggest the possibility of a subdural collection. Whenever suspicion arises regarding the presence of such a lesion the diagnostic issues should be clarified by multiple small cranial openings and/or ventriculography. Except under circumstances as described the surgical removal of a subdural blood clot during the first 24 to 48 hours following injury is of questionable value. Patients seriously injured should be given supportive treatment during the critical phase of their illness and surgical therapy instituted only after the vital signs indicate a degree of stabilization.

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VIRAL PNEUMONIAS*

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INTEREST in the form of pneumonia commonly called "Virus" pneumonia has increased rapidly in the past four years, and since the cause of the disease in the majority of cases is unknown, confusion in terminology has arisen. In the Army Medical Service, the disease is officially called "Primary Atypical Pneumonia, Etiology Unknown" to avoid error, since agents other than filterable viruses may cause a similar syndrome, and it is only surmised that a filterable virus is operative in most of the cases observed. The term is broad, however, and may include any pneumonia not conforming to the typical clinical lobar form. Even the popular term "virus" pneumonia is not specific. It is euphonic, but is as inclusive and as ungrammatic as the term "bacterium" pneumonia would be. The adjective "viral" is preferable. There are many kinds of bacterial pneumonias and perhaps as many of viral pneumonias, judging by the number and variety of filterable agents recently associated with them. Viral pneumonia is therefore a syndrome of which there are many causes. Reviews of the subject were recently published.[1,2] It is hoped that eventually, all of the causes will be discovered and simple diagnostic tests will be perfected to enable one to give specific names to the various viral pneumonias as in dealing with the bacterial ones. Some success in this direction has already been attained.

Several authors regard "Viral" pneumonia as a new disease, but the experience of others,[3] together with its diverse causation, certainly indicates that it is not. Descriptions of similar disease in medical publications decades old have been pointed out, [1,3b] To be sure, such descriptions can be found, but it seems futile to compare them with those of the present because of the emphasis then placed on clinical and pathologic aspects, in contrast with the current etiologic viewpoint. In retrospect it seems most likely that the viral pneumonias were considered as influenzal pneumonia, bronchopneumonia, migratory pneumonia, in-

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terstitial pneumonia[4] or capillary bronchiolitis, and that many cases were not recognized as pneumonia at all because of the paucity of physical signs and because roentgenograms were seldom made.

Another point was recently raised[5] as to whether or not "viral" pneumonia, as we now see it, is but a variant of the 1918 influenza, capable of assuming pandemic proportions and of clinical identity with pandemic influenza. It may be, of course, but in all reports thus far published there is a remarkable freedom from complications with pyogenic bacteria which played so fatal a role in the 1918 pandemic, and the mortality rate is nil in otherwise healthy persons. In fact, pneumococci of any type are seldom found in the nasopharynx during "viral" pneumonia, even less often than in healthy persons, 50 per cent of whom are said to be carriers of pneumococci.

Incidence: It may be only a coincidence that "viral" pneumonias sprang into prominence in 1938, after the introduction of sulfapyridine as a curative for pneumococcal pneumonia, but they have without doubt increased both in relative and in actual numbers since then. Several reasons may account for this, named in the order of probability: (1) Their actual increase may be a manifestation of the natural fluctuation in incidence common to many infectious diseases; (2) Diagnosis is made more frequently because of interest in the disease and because of the freer use of roentgenography; and (3) A diminution of the incidence of pneumococcal pneumonia induced naturally and also artificially with chemotherapy emphasizes a relative increase of unusual forms to which little attention has been paid in the past.

In our experience last year[2] "viral" pneumonias comprised about 15 per cent of the pneumonias admitted to the hospital; in a Detroit hospital[6] the proportion was as 6 is to 7, but thus far in the late months of 1942, "viral" pneumonias have outnumbered pneumococcal pneumonias on my service in the ratio of 3 to 1. It was thought that this increase occurred partly because pneumococcal pneumonia is now largely cured at home and that only patients with pneumonia refractory to chemotherapy come to the hospital. It has also been my impression that the undesirable custom of making diagnoses by exclusion is growing. Instead of troubling to determine the causal agent, chemotherapy is often used as a therapeutic test. In one report, the term "sulfonamide resistant pneumonia" was actually used.[7]

Etiology: The viruses of psittacosis, vaccinia, variola, measles and

influenza were long known to cause forms of viral pneumonia. More recently the viruses of varicella, lymphocytic choriomeningitis, a psittacosis-like disease (ornithosis) and other ones less well defined[8,9,10] have also been associated with the disease. Furthermore, the viruses of psittacosis and ornithosis have been found to be closely related to those of lymphogranuloma venereum and mouse meningopneumonitis, suggesting that some of these viruses may be widespread in birds and animals, which in turn may serve as sources of infection for Man. Several more viruses implicated in pneumonia have just been added to the list. A virus was isolated from sick cats belonging to a farmer's family afflicted with pneumonia. Evidence suggests that the same virus infected the human patients.[15] A similar circumstance was reported by Baker,[11] but in this case the causative virus was different, and preliminary studies strongly suggest its relation to the psittacine group. If relationship does exist and if the precedent of nomenclature for the psittacine group is valid, the name "alourosis" may be suggested for this infection in cats. The existence of this virus adds to the fascinating problem of epidemiology concerning the relationship and transference of viruses between birds, animals and Man. The question arises as to whether each of these apparently closely related viruses are descendants of a single parent form modified by adaptation in different hosts, whether one may change into another, if each represents a specific "type" of a genus, analogous with types of pneumococci, and each type selects the host in which it is best fitted to survive, or if they are identical. Indirect evidence already suggests that viruses of this group are implicated in from 15[12,16] to 50[2] per cent of cases of human "viral" pneumonias.

Eaton and his associates[13] recently succeeded in transmitting and establishing a virus presumably obtained from patients with viral pneumonia in the cotton rat. The evidence of a causal relationship of the viruses to the pneumonia in patients, however, is incomplete because of irregular results of neutralization tests.

General Forms of the Syndrome: According to my experience,[2] it would seem that the "viral" pneumonias fall into two general groups:

A. As a sporadic, non-seasonal, slightly contagious, systemic disease, with a relatively long incubation period of ten days to two weeks, occurring in isolated instances or in small groups of cases of varying severity, centering around a single source of infection. The epidemic disease in infants described by Adams probably falls into this classification.

In adults, the disease was at first confused[8] with psittacosis which it strongly resembles. It is probable that the viruses of the composite psittacine group of infections, lymphocytic choriomeningitis and the viruses established in the mongoose and in cotton rats among others yet unknown are operative. The disease appears to be a systemic one, often with splenomegaly and nervous symptoms, in which the lungs are incidentally affected. Manifestations of pulmonary involvement may be delayed for days. This feature has given rise to another undesirable term, "silent bronchopneumonia."[14] The upper part of the respiratory tract is seldom affected to the extent that it is in the other group. In the individual case, however, certain clinical signs and symptoms such as the normal or subnormal leukocyte count, the roentgenographic appearance of the lungs, sweating, unproductive paroxysmal cough, bradycardia, photophobia and the duration are the same as those in the next group.

B. As the severest cases in large epidemics of mild, highly contagious, local disease of the respiratory tract, commonly called colds, grip or influenza, occurring usually in the cold months. The incubation period appears to be short, a matter of one or several days. Generally the disease is indistinguishable from Influenza A or Influenza B, yet it is caused by a different agent or agents, and the three or more specific diseases often occur together in varying proportions in the same epidemic. It seems to be primarily a mild infection of the upper part of the respiratory tract which in the occasional case already affects the lungs or spreads downward to cause pneumonia and severe disease.

If general classification on this broad clinical or epidemiologic basis is correct, a tentative loose arrangement of the various causes of viral pneumonias may be listed as shown in Table I.

It is obvious that further progress in differentiation and classification awaits the discovery of the causative agents in whatever form the syndrome occurs.

Specific Therapy: All observers agree that the sulfonamide compounds have no favorable influence on viral pneumonias. In general, the signs, symptoms and results of laboratory tests of viral pneumonia are distinctive enough to permit quick differentiation from the usual forms of bacterial pneumonia, so that the practice of giving sulfonamides routinely to all patients with pneumonia regardless of the cause is unjustified. It also is unwise to make diagnoses by using sulfonamide compounds as a therapeutic test. In doubtful cases, or when evi-

TABLE I

Sporadic Non Seasonal Forms	*Epidemic, Winter Month Forms*
Varicella	Influenza A
Vaccinia	Influenza B
Variola	Measles
Psittacosis (parrot, parrakeet, etc. pneumonia)	"Cotton rat" virus(?)
Ornithosis (pigeon, chicken, etc. pneumonia)	Unknown varieties (colds, grip, etc.)
Ailourosis(?) (cat pneumonia)[11]	
Lymphocytic choriomeningitis	
"Mongoose" virus(?)	
"Cotton rat" virus(?)	
Feline pneumonia virus[15]	
Ill-defined[8,9] and unknown varieties	

dence suggests that bacteria sensitive to sulfonamide compounds have become secondary invaders, or if an epidemic of pneumococcal pneumonia is extant, or if pneumococci of the lower numbered types are present in the nasopharynx, chemotherapy is justified, but it should be discontinued if, in a reasonable period, the disease fails to respond.

Convalescent serum from patients who have recently recovered from a viral pneumonia has also been used in therapy and a few clinicians claim to have observed beneficial effects. In view of the diverse causes of the syndrome, this method of treatment seems to be empirical to say the least. Even specific convalescent serum once thought to be helpful in psittacosis, has long since been given up as useless.

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THE NATURE OF PSYCHOTHERAPY*

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PSYCHOTHERAPY is as old as medicine: indeed, it is nearly as old as religion; and a history of psychotherapy would be a history of human culture. Yet it is only the last half century that has brought scientific knowledge of its scope and limitations; and it is only in the last half century that anything new has been added to its techniques.

In the perspective of history, the term itself is young. Yet it has already acquired a meaning so vague and general as to be difficult to use with precision. It can include the mystical healing rites of a priest-physician of ancient Greece, or the drum-beating and the voodoo practices of a modern primitive, David strumming his lyre against Saul's melancholy or classes in rhythmic dancing in a modern psychiatric clinic, forced labor in an old prison asylum or the rhythm of a modern occupational class in basket weaving. As one witty patient described it, "under one, and over two, rest in a neutral environment."

In this loose sense, psychotherapy embraces any effort to influence human thought or feeling or conduct, by precept or by example, by wit or humor, by exhortation or appeals to reason, by distraction or diversion, by rewards or punishments, by charity or social service, by education, or by the contagion of another's spirit. This broadest possible use of the term would also include the temporary lift of spirit through music, art, or literature.

Such methods as these depend upon an artful blend of imagination, feeling, intuition, firmness and common sense; but it will be our thesis that as a science psychotherapy begins only where these leave off. It happens that it usually will be good common sense to give water to a thirsty man; but this is not science until we know the role of water both in the normal body and in its pathology. So too with all such simple psychotherapeutic sips of water as sympathy or advice or rewards and punishments: we use these with scientific precision only when we

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know their relationship to normal and disturbed functions of the mind. Therefore, our first task is to formulate a concept of psychological normality and to define the boundary line between normality and neurosis.

We will not be aided in this by any distinction between normal and neurotic which is based on the usual differential points. We are not concerned with the issue of whether a man knows the difference between right and wrong in the archaic and unrealistic legal sense, nor with the question of whether he believes that he knows what he is doing and why he is doing it, that is, whether he can rationalize his conduct. Nor are we concerned whether his conduct conforms to the social and cultural mores of any special time and place, or whether it is extravagant and fantastic or orderly and sedate. These are important aspects of behavior but not basic or constant in their implications. Thus it is normal to wash one's hands but not normal to have a handwashing compulsion, although there is nothing eccentric or anti-social or deviant from any culture in the mere act of handwashing. Similarly, to stand on your head in a tumbling act is normal, while to stand on your head in church is not; unless perhaps it is done to pay an election bet, or as a hazing stunt. Evidently the critical difference must be sought neither in the act nor in its setting, but in the determining mechanisms of the act. Behavior is normal or abnormal because of the nature of the inner forces which produce it.

What then are the characteristics of the inner determining forces which make any moment of thought, feeling, and action normal, and what are the characteristics which make it neurotic. The answer to this requires one further distinction—a distinction between forces which are within the range of our conscious perceptions, and those which are so deeply buried as to be inaccessible to any introspective knowledge of ourselves. It is as difficult for men who are unfamiliar with this field of phenomena to realize that unconscious yet psychologically powerful forces are constantly at work in everyone, as it was for the old scientist who had never looked through a microscope to believe in the microscopic objects which von Loevenhoeck described. Nevertheless it is one of the great achievements of modern science that the evidence for this has become incontrovertible. It is not possible to describe this evidence in detail; but it is important to note in passing that it is firmly based on a variety of different techniques applied to four fields of observation: to wit, experimental work with humans, clinical observations with psy-

choanalytic methods, clinical observation of animal behavior, and experimental work with the conditioned reflex. The weight of this varied evidence forces the psychiatrist to proceed from the premise that in every moment of human life the mosaic of thought, feeling and conduct which constitutes behavior is determined by confluent forces, that we can be aware of only a fraction of these forces at any time, and that we tend naively to overestimate the relative power of those of which we are aware even in events in which the unrecognized forces have played a determining role. To simplify our terminology, we can use a somewhat inaccurate foreshortened jargon and say that while we are conscious at any moment of some of the reasons for our conduct we are always unconscious of others.

But how does this help us understand the distinction between the normal and abnormal in behavior? Here we are on clear grounds. Whether "conscious" or "unconscious" forces play a major part in producing any moment of behavior is the most important single distinction in human psychology. This is because conscious motives are constantly molded by external realities, whereas unconscious forces pay no heed to the outside world. They are uninfluenced equally by the pain that they may cause the individual himself and by the pain which they may inflict on others. They are deaf to exhortation, argument, and reason, or to appeals to love or hate. They are so blind that the symptomatic acts which they engender do not succeed in satisfying even themselves. Their tensions remain undischarged. Therefore they are literally insatiable. Consequently, the larger is their influence in any particular act, the more unrealistic do they make that act, and the more inflexible and the more endlessly repetitive it becomes. This is what has been called the "Repetitive Core of Neurosis," Kubie:[1] and this is why neurotic behavior is endlessly and ceaselessly repetitive, whether it appears in the form of overt neurotic symptoms or of neurotic life patterns disguised as ordinary behavior. This is what the layman means when he says that we learn so little by experience. It would be more accurate to say that the consciously organized normal aspects of man can learn by experience, but not that part of him which is unconsciously driven: because the unconscious neurotic mechanisms go on their way unsated by gratification and unaltered by experience.

It is precisely this repetitiveness which constitutes the essential challenge to psychotherapy and its most difficult problem. However, the

approach to this ultimate stronghold is always preceded by less complicated preparatory maneuvers. These simpler psychotherapeutic expedients may be grouped under three main headings: (a) Practical support—consisting primarily of advice, guidance, and assistance in the management of life situations and environmental difficulties through social service aids, etc. (b) Emotional support—consisting essentially of sympathy, exhortation, admonition, encouragement, humor, art, recreation, companionship, etc. (c) Reorienting education—consisting primarily of efforts to alter the patient's habitual attitudes of guilt, fear, hate, and depression, by educating him to tolerate his own conscious and unconscious needs and cravings, his instinctual hungers, his familial jealousies and hates, etc.

The third of these groups requires extensive knowledge of unconscious psychological forces, and hides many subtle dangers which will be discussed below. The first two, however, can hardly be called a discovery of the psychiatrist. They are the homely non-specific, commonsense weapons of every wise parent and teacher. They must always be tried first. Indeed where they succeed alone, one may be sure that the maladjustment is more external than internal: since it is precisely their failure which reveals the rigid repetitiveness of behavior which is energized predominantly by unconscious internal forces, rather than by external environmental stresses. This again is why we say that psychotherapy as a science begins where the simple, rational methods fail.

Perhaps a banal example can make this clear. A child of three or four wakens at night, slips out of his bed, and comes pattering into the front room in search of his parents. On the first occasion he is picked up, hugged, and carried back to his room; and after his wants are attended to he is tucked in, kissed and left there. A few minutes later, however, he reappears. The same "medicine" is applied, perhaps a little more brusquely, but nonetheless kindly. Even if it didn't work the first time it might work now. But again the youngster appears. The parents try successively leaving the light on or the door open, a toy in his bed, cuddling, promises, bribes, scoldings, threats or punishments; but in spite of their versatile efforts the child appears over and over through the course of many evenings. This is a typical transient neurotic episode of childhood. The parents become distracted. They run through the whole gamut of human feelings, from affectionate patience to irritability, anger, and finally a panicky feeling that something is wrong.

They feel that they have tried every possible sensible device, without success. They do not realize that their failure constitutes a thorough therapeutic test, demonstrating that the roots of the child's disturbed behavior lie deeply buried, on a level which is inaccessible to any surface manipulations.

What is true of the small child is also true of the adult. Rewards and punishments, discipline and education, argument and reason, the sensible manipulation of the environment, occupational therapy, comfort, solace and exhortation, these are all essential preliminary steps in the approach to any psychotherapeutic problem. They clear away external complications. They effect cures when the situation is sick rather than the individual. Thus they are at once the ante-chamber to deeper treatment, and a therapeutic test of its necessity. Where they work it is folly to go deeper: but where they fail it is even greater folly to persist with education, habit training, social service, work adjustment, etc., because, until he is already on the road to health, the neurotic patient cannot use such aids, no matter how badly he needs and craves them. For the neurotic, treatment must precede practical assistance: or the wisest guidance will be sabotaged by the neurosis.

But psychotherapy cannot always achieve or even aim at the eradication of causes. As in any other medical discipline, it must sometimes content itself with palliative measures.

Palliative psychotherapy consists primarily of an effort to teach patients how to live with some measure of comfort within the confines of their uncured neuroses. Of itself this can relieve much suffering. For instance, an agoraphobia may manifest itself first merely in an uneasy feeling in very large, open spaces. At this stage the neurosis will make little difference in a patient's life. Gradually, however, the anxiety will become more all-pervading, until the patient becomes unable to go out of doors at all, or even to go into a moderately large room. With each successive encroachment on his freedom, his life and that of his family become more circumscribed. Soon all who know him begin to pay with him the price of his neurosis. If the psychotherapist does no more than to keep the patient from succumbing to these successive restrictions, he will contribute greatly to the happiness of the patient and of his family. It is here that education, exhortation and endless ingenious manipulations of the environment can play a role of major importance.

If more than palliative treatment is to be attempted, the psychothera-

pist must attack the rigid, inflexible, automatic, repetitive core of the neurotic process. It will be seen that this challenge cannot be effectively met in the naive spirit which underlies the time-worn concept of "habit training."

It is not orthodox to place this rigid repetitive tendency at the very heart of our psychotherapeutic problem: yet I do so because I believe that this is the nucleus which is common to all neurotic processes, and that the customary emphasis on mood and content is an emphasis on secondary symptomatic manifestations. When the repetitive process manifests itself in certain recurrent ideas we speak of obsessions, in recurrent acts we speak of compulsions, in recurrent mood states we speak of anxieties, depressions, elations, etc., and in recurrent distortions in the patient's view of external reality we speak of delusions. These variegated secondary symptoms demand flexible temporizing care: but they are the branches and not the roots of the tree. It is towards the roots, i.e., the forces that make the disturbance recur, that our therapeutic efforts must be directed.

First, however, we must raise one more important preliminary question, namely, whether the repetitive pattern ever becomes so structuralized in the nervous and glandular organization of the individual as to be unalterable by experience on the psychological level; or whether the mechanism by which that force is mediated can be altered by means of psychological events. On the answer to this question hinges the future of all psychotherapy: yet the answer is not known. We know that the duration of a symptom is no indication as to its structuralization. For instance, as a result of purely psychological treatment I have seen a man of 57 lose symptoms which began when he was three years and ten months old: while in other cases, in spite of penetrating insight into buried forces, quite recent symptomatic manifestations may persist almost unaltered. We do not yet know why this is true. It is possible that in certain patients the nuclear repetitive phenomena may be the expression of an inherent organic inclination towards such repetitions. We know that repetitive phenomena occur universally and from infancy onward; but as yet no quantitative studies are possible of their incidence, of the factors which produce them, or of their correlation with later neurotic manifestations. In brain injuries and in certain experimental lesions repetitive phenomena may be produced; and Brickner[2] has shown that the stimulation of a certain area in the brain can induce persevera-

tion. This does no more than suggest the possibility that organic factors may play a role. It gives no information as to possible variations in the inherent inclinations of different brains towards the production of repetitive states under similar psychological stresses. Therefore, we are left with several alternative possibilities, which are not mutually exclusive. In the first place, there may be a greater inherent inclination towards stereotyped repetitive phenomena in one brain than in another. Secondly, certain types of gross or subtle injury to the brain may possibly enhance this tendency. Thirdly, psychological influences of varying severity can induce this phenomenon. Presumably if sufficiently intense they can induce the phenomenon in any central nervous system: but it is also possible, if not probable, that in certain nervous systems they can be induced more readily than in others, and that once they arise in certain brains they may thereafter be more tenacious than in others. Finally, repetitive phenomena once induced may be mediated by reversible changes in one nervous system, and by irreversible or even progressive changes in another. It is particularly in such cases as this that lobotomy may be a rational procedure. Our knowledge of all of these matters waits upon the development of methods by which these hypothetical variables can be measured. In the absence of such methods in our pursuit of therapeutic weapons we are forced to rely upon the oldest device in medicine, the method of trial and error. Let us turn to the errors first, to dispose of them.

Throughout his life every psychiatrist learns one lesson over and over again, only to forget it, namely, that however much it may relieve his own feelings it does no good to a patient to call his illness names. When a patient comes with a frank symptom, such as a hand-washing compulsion, we know that it is a mere waste of breath to tell him solemnly that he suffers from a compulsion. The patient tells us that himself. Our technical nomenclature can help him neither to control his symptom nor to get rid of it. Nevertheless, when dealing with subtler compulsive patterns this is forgotten, and psychiatrists hopefully describe to patients their "neurotic trends." There is no parent who in layman's terms has not told his child the same thing, calling him lazy or good-for-nothing or disobedient. Scientific name-calling is not more effective than this: because scientific epithets can not alter the repetitive nucleus of a single neurotic manifestation. At worst they merely mobilize the patient's defenses. Jung and Adler and Rank all made that mis-

take. It is regrettable to see it recurrently repeated by others. Many years ago William Allen White pleaded against this error, when he said that the hardest lesson a psychiatrist has to learn is to allow a patient to say that 2 plus 2 equals 5, until the patient discovers why he *had to* cling to this belief.

It is impossible to lay too much emphasis on the futility and dangers of naive confrontation; because this is the most frequent mistake of the immature psychiatrist, and of the well-intentioned general practitioner who has read psychiatric theory without having rigorous drill in its procedures. Superficial confrontations which merely describe the patient to himself without long and patient preparation, inevitably precipitate resentment and mobilize his defenses. Premature confrontations which reach to deeper levels, challenging the patient to recognize some of his deeper fears, yearnings and hates, must either be rejected as far-fetched or else they will precipitate the patient into unmanageable terror, guilt or depression. One learns slowly never to force a patient to look at a painful fact about himself until you are in a position to explain its origin in his history and its function in his present life. This may take patient exploration over many months.

So important is this that we must recognize that it may be dangerous to attempt to argue a patient out of cherished ideas and beliefs even about seemingly impersonal affairs, such as science or politics. Argument may be as dangerous as confrontation. If it succeeds in breaking down the defensive barrier of symptomatic ideas, all that can be achieved is a sudden eruption of overpowering emotions.

Thirdly, there may be danger even in reassurance or consolation. A patient said a few mornings ago, "I was in a panic. My sister telephoned me and assured me that her household were well. Then all of a sudden I found myself in such a depression that I stood at the window and thought of throwing myself out." It is not too much to say that the only safe thing to do with a patient is to listen to him, until one understands enough about him to be in a position to relate everything one says and does to *his* unconscious psychological processes.

And now we are ready at last to suggest a possible answer to the final question: namely, what is the nature of a psychological process that can influence the basic underlying repetitive patterns which characterize all neurotic phenomena? This may not be too difficult to understand if we recall that the repetitive pattern is a recurring effort to bring to a

satisfactory completion a painful, forgotten, and incomplete experience which has been left hanging in air. Thus the child who wakens in the midst of a nightmare puts himself to sleep again by telling himself a happy ending. The man who has been worsted in an altercation goes down the street muttering all the fine things that he might have said had he only thought of them in time. Indeed we do this throughout life, the only difference being that most of our attempts to discharge the tensions left by past experiences are carried through without our knowing what we are doing.

In such experiences, moreover, the boundary line between reality and dream is never hard and fast, because each "experience" is a mosaic of fact, confusion, misunderstanding, and fantasy. The past which we try vainly to undo is half real and half imaginary: but the neurotic shadows which it casts on the present screen of life are real enough. The neurotic component in all of us is always the residue of just such incomplete experiences, nightmares which have not lost their emotional hold even after we have wakened, nor after we have forgotten the dreams themselves. It is as though we had wakened just as the villain was poised to strike. Then we "forget" the dream but not the feeling, and live out our waking lives through symptoms or symptomatic acts which are disguised and unsuccessful attempts to avert or avenge the fancied blow.

One characteristic form of the acute traumatic war neurosis gives us a clear picture of this process. Night afer night the patient attempts self-cure. That is, he redreams his shattering experience, trying each time to dream it through to a happy ending. But as he dreams, his terror mounts to a point at which he is wakened prematurely at the very climax of danger. This leaves him shaken with terror, grief and rage. There is a point in the dream beyond which he cannot go, some mingling of reality and fantasy so highly charged that every time he tries to re-live it his mind shies away at the last moment like a horse that refuses just before the take-off. Just as this happens spontaneously in the war neurosis, so it occurs in the treatment of the neurotic patient. He, too, must shy away from deeply painful if less melodramatic early experiences, whenever the therapist tries to help him re-live them to a realistic conclusion, thus to free him from the endless, closed repetitive circles of his neurotic patterns.

This incessant struggle is the source of a state of constant inner tension, for which the most obvious therapy would be a discharge of the

pent-up feelings. The process of discharge is often called "catharsis," and much is made of it in psychiatric literature. Therefore, it is important to know that catharsis is not enough. Furthermore, the concept is poorly understood and widely misused. Any discharge of emotional tension can give temporary relief, be it a temper tantrum or an uncontrolled outburst of crying and laughing. Even a painful symptom gives momentary relief. The patient with a compulsion neurosis discharges tension in the moment in which he executes his compulsion. In fact, every act and thought and feeling discharges tension in some measure; and all of this is catharsis. Unfortunately, however, most of these acts rapidly recreate as much tension as they discharge, and sometimes more. In a somewhat more lasting sense, however, it is cathartic to help a patient to express pent-up feelings which he has previously been afraid or ashamed to face. A man who has never been ready to admit that he hated some member of his own family may find marked relief when the psychiatrist's non-critical attitude encourages him to acknowledge this feeling. But even here, a fresh up-welling of guilt or anxiety can exact a heavy price for the catharsis, and precipitate the patient into graver depression and terror. Thus, no mere discharge of feeling, either from the surface or from the depths, can accomplish lasting benefits unless it is preceded and accompanied by an eradication of the source of the feelings. Without this, the feelings inevitably recur.

A psychotherapy which aims at the eradication of etiological sources must recover the content of the forgotten dreamlike "experiences," since it is these which give rise to the feelings which produce symptoms. They are the links in the chain of conditioning experiences. Therapy must complete the dream, in order to dispel the haunting moods which flow from it. It must find the happy ending which the child in us seeks incessantly. And in order to recover that which was forgotten it must penetrate the mists which shroud the past, and enable the patient to relive vividly the acutely painful and confused experiences which make up the "dream."

All of the defensive forces of the patient's personality are mobilized against any such effort to penetrate into the forgotten areas of experience, because they have been "forgotten" not by accident, but as an obligatory defense against the pain of the original experience. Therefore, every technical advance in psychotherapy has been in the direction of finding ways around these defensive barriers.

This whole matter is widely misunderstood. The question is asked, "Does the rediscovery of past events automatically dissipate feelings which had their roots in those particular events?" Or in another form the question is asked, "Does the discovery of a secret purpose, yearning, or desire automatically dissipate that purpose?" In reality these are not two questions but one. It is not merely the recovery of an event which releases the patient, nor merely the recovery of the event plus the feelings and desires which derive from that event. It is the discovery of the totality of the purposes, the hopes, the fears, the loves, and the hates which animated the individual at the moment of the event, plus what that event did to those purposes, loves and fears and hates, and how these were deviated by that event from their initial pathway onto another. This, and not less than this, is the potent discovery. If at a certain moment in the life of an individual the bank of a river assumed a terror-laden significance and if that moment and all of its attendant circumstances were subsequently "forgotten," then the terror becomes isolated, like a station with no railroad tracks leading to or from it. The terror can be experienced but only about substitutes; and can never be discharged. And the recovery of the memory of the moment when for the first time the bank of the river acquired a nightmarish quality allows the whole structure to be dissipated, until the bank of the river again becomes only a bank of a river and nothing more. The recovery of facts thus is merely a necessary part of the total process of undoing experience, of deconditioning the human animal. This is why "habit training" is a naive and empty concept.

All highly charged symptoms acquire many meanings through the course of life. Consequently, in retrospect one must trace its evolution through a series of discoveries, each of which gives temporary relief and then a disappointing recurrence of pain. This has led some theorists to the easy conclusion that the discovery of the genetic experiences is unimportant. This is equivalent to saying that it is unimportant to discover the bacterial organism which causes an infection if you cannot always prepare a powerful antitoxin from it.

A full consideration of the methods of recovering the past would involve us in difficult technical details concerning the handling of various types of resistances and transference phenomena. It is not possible to do justice to these problems here; but it may be useful to emphasize that the strength and power of the patient's defenses constitute the final

reason why blunt confrontation is worse than useless, usually serving merely to increase the patient's opposition, and throw him into panic, and from panic into rage. In turn, this is why all short-cuts are dangerous.

During recent years various methods have been developed by which this goal is pursued. The most important of these is psychoanalysis; not because it is a perfect instrument, but because it is still the pathfinder. Hypnotism, which once was used naively as a magic wand with which to order symptoms to disappear, has more recently been used as a method to assist in the recovery and re-living of the past. It is used both in combination with and as an extension of psychoanalytic technique. In turn, both of these have been used in combination with medications of various kinds; and experiments are under way in their use in combination with hypnagogic states. Finally, even the various forms of shock treatment come into consideration in this connection, because they sometimes render material accessible which is otherwise hidden behind the repetitive patterns of thought and behavior. Shock treatment can be used, therefore, not merely as a crude way of disrupting temporarily the associative pathways, but also to render accessible to investigation the life experiences which lie buried behind those patterns.

Not one of these methods has as yet established beyond argument its own territory of assured pre-eminence, nor its own limitations. Each is in the developing, testing and formative stage. Few psychiatrists would agree to any formulation of the indications or contraindications of any one of them. Under these circumstances, little would be gained by putting forward my own views of the moment: especially since they, too, will undoubtedly change with the experiences of tomorrow. However, if we have succeeded in making clear the basic principle and goal which underly all of them, and if we have brought the concept of psychotherapy into harmony with scientific medical attitudes on the relationship of therapy to etiology, then I will feel that the main purpose of this paper has been achieved.

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THE DIAGNOSIS OF CANCER OF THE PROSTATE INCLUDING THE INTERPRETATION OF SERUM PHOSPHATASE VALUES*

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In the philosophy of diagnosis, two medical aphorisms must be openly or tacitly assumed, both of which apply to prostatic cancer. R. C. Cabot frequently stated the first; that the symptoms of any disease may be none. The second consideration follows: in order to make a diagnosis one first must have the disease in mind. The clinical picture of some diseases is so apparent as to compel recognition, as in complete urinary retention from prostatic cancer; or the manifestations of disease may be highly subtle and obscure, as when metastasis of a prostatic tumor produces chest pain or sciatica, the primary lesion being asymptomatic.

The diagnosis of prostatic cancer is frequently satisfactory since chemical methods exist by which the disease often may be recognized in the laboratory. The final aim of diagnosis is to recognize disease by chemical methods without seeing the patient. Laboratory determination of "What Label" and its yardstick "How Much" is of first importance in classification, in evaluating therapy and even in initiating new forms of treatment. In prostatic cancer, laboratory testing of blood from the veins of the antecubital fossa is of significance.

Cancer of the prostate is the cause of death of about 5 per cent of men over fifty years. The diagnosis of clinically active prostatic cancer often is easy since usually the lesion is far advanced and has metastasized when the patient presents himself for treatment. Clinically active prostatic cancer, in any early stage suitable for total excision of the entire gland, usually can be recognized provided palpation of the prostate *per rectum* is carried out; such early lesions, however, are not often detected at present. An interesting observation of the pathologists is the frequent presence of occult, miniature prostatic cancers which do not cause mor-

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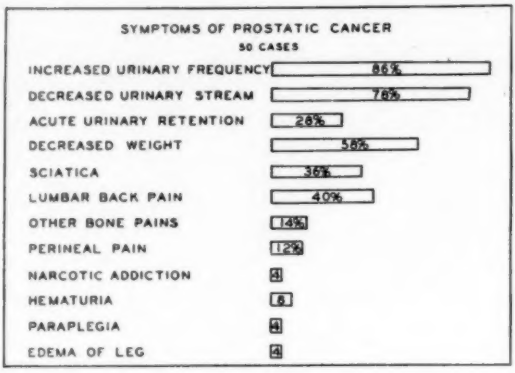

TABLE I

bidity; the incidence of these lesions in the prostate gland of men over 50 years of age examined routinely at autopsy, ranged from 14 per cent (Rich), 16.7 per cent (Moore) to 46 per cent (Baron and Angrist[1]). It is clear that the diagnosis of these small silent lesions cannot be made except by guess on a statistical basis, and indeed it is perhaps of little clinical importance; however, in view of the stimulating effect of male sex hormones on prostatic cancer, it is well to bear in mind that these small occult lesions exist with frequency in older men, when these hormones are administered to men over fifty years of age.

Symptoms of Prostatic Cancer: A typical history for a run of the mine case of prostatic cancer is that of a man over fifty years of age who has increased frequency and some difficulty on urination, with pain in the lower back and loss of weight.

The usual symptoms of prostatic cancer depend on interference with micturition by the primary tumor, or by osseous or lymphatic metastasis; or as a result of the cachexia of carcinomatosis. It is uncommon for the metastases of prostatic cancer to produce important symptoms except through involvement of bones or the regional lymph nodes. Nearly all patients have increased urinary frequency and difficulty in voiding (Table 1); hematuria usually is not present. In our experience 56 per cent of the cases suffered from pains in the bones, the result of metastasis. Most commonly these symptoms are in the lumbar region or legs; they may arise in any other part of the spine with radicular radiation.

The age incidence of prostatic cancer is of interest; the disease is essentially that of men over fifty years, and we have seen only one case in a person (aged forty-seven years) younger than this age.

Physical Findings in Prostatic Cancer: The characteristic findings are obtained on rectal examination and consist of a stony hard, nodular, craggy involvement anywhere in the prostate. In early disease, the nodule is single; in later disease multiple nodules with infiltration along the seminal vehicles are present. It is very rare for prostatic cancer to ulcerate into the rectum; most of the ulcerative neoplasms on the anterior rectal wall in the prostatic region are rectal tumors.

Single hard nodules, not caused by carcinoma, have been encountered occasionally in our experience, and have been due to prostatic calculi, tuberculosis, non-specific infection, as well as benign prostatic hypertrophy, which is usually the most puzzling condition to be considered in differential diagnosis. Roentgenologic examination of the pelvis should always be made in suspected or definite prostatic malignancy and will demonstrate prostatic calculi if they are the cause of nodule formation.

Roentgenologic Findings in Prostatic Cancer: In a series of fifty patients with advanced prostatic cancer, x-ray evidence of osseous metastasis was present in thirty-one cases. Both osteoblastic and osteolytic types are found; usually they occur together. Only in two cases were osteolytic lesions alone encountered; these lytic lesions are commonly small and multiple compared with the large "melting away" that is often seen in hypernephroma. The pelvis was always involved when metastases had occurred in the bones, and lesions were never encountered distal to the knee or elbow joints. Other bones are often involved, but as a routine measure of search for metastases, raying of the pelvis alone is regarded as an adequate device. In the improvement which occurs following endocrine treatment, osteolytic lesions become sclerotic.

The chief confusing entity in x-ray diagnosis of prostatic metastases to bone occurs with Paget's disease (osteitis deformans). The fundamental x-ray evidence of Paget's disease is an increased grossness of trabeculae with cyst formation; the trabeculae become massive. In prostatic cancer the trabeculae of bone disappear, incorporated in dense osteosclerotic masses.

Pathologic Diagnosis of Prostatic Cancer: It is occasionally necessary to resort to biopsy to make the diagnosis of cancer of the prostate. Several methods of approach are available and each of these has definite

indications. The perineal approach with removal of a section of the gland, is best done in suspected early cases, where radical prostatectomy can be carried out immediately if the diagnosis is confirmed. If there is prostatic obstruction in advanced cancer, transurethral resection of the prostate should be carried out since in addition to obtaining tissue for biopsy the obstruction may be removed effectively. The author has had no experience with the valuable method of aspiration biopsy which has been developed at Memorial Hospital.

The essential criteria for pathologic diagnosis are anaplasia and invasion. The relation of the small acini to the surrounding stroma in adenocarcinoma is of importance.

In differentiating carcinoma primary in the prostate from bladder carcinoma or other malignant disease in this neighborhood, valuable aid is often obtained by staining the tissue for acid phosphatase by the technique of Gomori, since prostatic epithelium contains much larger amounts of this enzyme than other tissues. The tissue requires special fixation in acetone preliminary to staining for the sites of enzymatic action.

Cystoscopic Diagnosis of Prostatic Cancer: When prostatic cancer is associated with benign prostatic hypertrophy, as not infrequently occurs, only the benign lesion is visible at cystoscopic examination. Unaccompanied by hypertrophy, advanced prostatic cancer often displays suggestive features; such as constriction of the posterior urethra and infiltration of the trigone. A common sign is the appearance of small irregular notches around the vesical neck compared with the notches found in prostatic hypertrophy which resemble formal architectural figures, Gothic in type.

Serologic Diagnosis of Prostatic Cancer: This important diagnostic procedure was developed by A. B. and E. B. Gutman[2,3] of Columbia University and Barringer and Woodward[4] of Memorial Hospital independently in 1938 and depends on the important discovery of Gutman, Sproul and Gutman (1936)[5] that prostatic cancer contains large amounts of acid phosphatase. In brief, there exist two distinct enzymes, acid and alkaline phosphatase, which are capable of splitting monophosphoric esters to form inorganic P.. Alkaline phosphatase is particularly rich in concentration in the osteoblasts and when there is brisk osteoblastic activity the value of this enzyme is increased in the blood serum; such states include rapid growth in children, Paget's disease, osteogenic sar-

coma, osteoblastic metastasis from prostatic cancer, etc. This enzyme is also increased in liver disease where it is an exquisite indicator of hepatic function. So far as is known, bone activity and hepatopathy are the only conditions in which the serum values are increased.

Acid phosphatase values of serum are significantly elevated only in cancer of the prostate and in this condition only when metastasis has occurred. In the author's laboratory[6,7,8] the King and Armstrong method, with appropriate buffering, is used for determination of both enzymes; the normal values are, for acid phosphatase, less than 4.5 units; and for alkaline phosphatase, less than 12.5 units, each per 100 cc. of serum. In a series of 100 cases of advanced prostatic cancer before treatment, one or both of the phosphatases were increased in 52 per cent, and both were in the normal range in 48 per cent; with osteolytic metastasis alone the phosphatases were never increased. Usually when there is an elevation of phosphatases, both are increased; however, in one case only the acid value was high and in five cases only alkaline phosphatase was elevated. When the serum acid phosphatase is significantly raised (greater than 10 units) it is certain that the patient has prostatic cancer with metastasis; values between 5 and 10 units are presumptive of prostatic cancer, but are occasionally observed in other conditions. Patients frequently have advanced prostatic cancer even with metastasis to the bones accompanied with serum phosphatases in the normal range, but if both phosphatases are distinctly increased the diagnosis is established that cancer of the prostate with osteosclerotic osseous metastasis is present; there are false negatives, but no false positives. These tests are of special value when roentgenograms of the bones show equivocal changes. It is obviously necessary to determine both of the phosphatases for maximum efficiency in diagnosis and prognosis of cancer of the prostate; these tests are as clearly necessary as studies of basal metabolism in thyroid disease.

Prognostic Implications of the Serum Phosphatases in Prostatic Cancer: I believe that it has now been established that the prostatic cancer cells flourish in the presence of physiologically effective amounts of male sex hormones, and often retrogress, more or less, when the testicular androgens are eliminated through bilateral orchiectomy or are neutralized through estrogen administration. The course of the disease may be followed well by frequent serial phosphatase determinations. Characteristically when subjected to hormonal modifications in those patients with

phosphatase elevation, the acid phosphatase level rapidly falls after initiating treatment, while the alkaline phosphatase values rise for some weeks (a favorable sign) and then fall to or towards normal. In some patients the values having reached normal have remained so for at least 800 days following orchiectomy.

In one patient with complete paraplegia resulting from metastatic cancer of the prostate, the alkaline phosphatases precipitously fell to normal in the few weeks in which the paralysis was disappearing following orchiectomy; this fall, not preceded by the characteristic rise was attributed to the bony atrophy of disuse accompanying the high paraplegia. When the phosphatases do not return to normal, following endocrine modification, the prognosis is unfavorable. In a group of patients in whom the clinical and chemical response to this treatment is favorable, after some months alkaline phosphatase values again climb accompanied by clinical evidence of activation of the disease: this enzymatic response is likewise unfavorable.

Certainly, frequent serial phosphatase determinations in prostatic cancer have passed from the stage of being merely an important laboratory tool to an essential clinical status in the diagnosis and prognosis of cancer of the prostate.

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THE MANAGEMENT OF THE ACUTE EPISODE IN CORONARY OCCLUSION*

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RECENTLY the United States Census Bureau[1] reported that the death rate from heart diseases has more than doubled during the last 40 years. Deaths from heart diseases in 1940 totaled 385,191, a rate of 292.5 per 100,000 population. This was the greatest number of deaths ever recorded from heart diseases.

Although there have been appreciable decreases in heart disease death rates in the low age brackets, the rate of heart disease fatalities has increased considerably in the upper age groups; for example, 45 to 54 years from 173.01 to 279.5; 55 to 64 years from 414.1 to 713.5; from 65 to 74 years from 957.3 to 1,723.5. Most of the deaths in these groups were due to coronary artery disease or its complications including coronary occlusion and myocardial infarction. This gives some idea of the prevalence of the condition, treatment of which I have been assigned to discuss this afternoon.

Definition of terms: The title of this discussion would probably be more correct if myocardial infarction were used in place of coronary occlusion. Recently some confusion has arisen concerning the terminology in coronary artery disease, due probably to several new expressions suggested such as coronary occlusion, coronary failure and coronary insufficiency. The term acute myocardial infarction seems to fulfill the requirements since the clinical signs and symptoms are all on the basis of myocardial necrosis. Then too, it is known that this lesion may occur without coronary thrombosis or without acute coronary occlusion, and contrariwise, occlusion or thrombosis of a coronary artery may occur without producing a myocardial infarct. As the studies of Blumgart and Schlesinger indicate, there is no characteristic clinical syndrome associated with coronary artery occlusion per se. The symptoma-

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tology is usually the result of an infarct.

Arbitrarily the first three weeks were chosen as the phase of myocardial infarction to be considered in this talk. An analysis of some 200 cases of myocardial infarction[2] on the Third (New York University) Medical Division of Bellevue Hospital demonstrated that most of the complications and sudden deaths occur during the three weeks after the onset of the illness. It is obvious, therefore, that this is the critical period of the disease.

Diagnosis: In the typical case of myocardial infarction the diagnosis is easy, in fact, is often apparent from the history alone. Although the problem of diagnosis will not be discussed, I should like to stress the importance of early recognition and the institution of immediate treatment since they have a decided influence on mortality as well as future expectancy of life. As already mentioned the early phase of this disease is usually the critical period. In a patient in whom a diagnosis is not obvious bedrest should be maintained until the correct diagnosis is decided upon and thereby give the patient the benefit of the doubt. The subsequent appearance of additional clinical manifestations indicative of infarction and the alterations in repeated electrocardiograms will usually establish the true nature of the disease even in the bizarre or atypical cases.

Treatment: Thrombosis which is the precipitating factor in some 60 per cent of cases with myocardial infarction is not instantaneous and may extend over a period of hours, days or weeks. Premonitory symptoms in such instances may precede the acute episode of infarction over the same period of time. It has been suggested that heparin, and more recently dicoumarin[3] (dicumarol), the new anticoagulant, might be useful in such cases in preventing complete obstruction and, in turn, infarction. Experimentally the basis for this rests in the work of Best and his collaborators[4,5] in dogs in whom heparin prevented the thrombosis which usually occurs in their coronary arteries after sodium ricinoleate has been injected into these vessels. This suggested the possibility of its use clinically in the initial stages of coronary thrombosis; or, if thrombosis be already complete, heparin might arrest its extension, or prevent mural thrombosis which not infrequently occurs in the ventricles over the site of the infarct and which acts as a source of emboli.

I have failed to find any reference in the literature relating to such use of heparin. Best[5] states that "it has been used in a few cases of coron-

ary thrombosis in human beings but not as yet in a scientific manner." Several isolated cases apparently have been known to have been reported at various meetings but these have not as yet been published.

As to dicoumarin (dicumarol) the new anticoagulant agent isolated from spoiled sweet clover, I have just been informed that it has been employed recently in four cases of coronary thrombosis, one under the supervision of Duryee[6] at the New York Post-Graduate Hospital and the other three at Welfare Hospital under Prandoni.[6] In all four instances it was administered in doses of 100 mgm. per day by mouth over periods ranging from twenty-one to twenty-eight days. All four patients had uneventful courses and recovered.

It is Best's[7] feeling, and I agree heartily with him, that the results in a few cases in which any anticoagulants have been employed are not significant, and that until a series of such cases with adequate controls have been made available, it is rather useless to discuss the results of the use of anticoagulants in the treatment of clinical coronary disease.

As soon as the diagnosis has been established every effort should be made to obtain complete physical and mental rest for the patient. He should be put to bed immediately, preferably in a Gatch bed, and not moved from it for at least a month. If transportation is necessary it should be by means of an ambulance and, if possible, postponed until at least the end of the third week. Under no circumstances should he be moved to a hospital merely to obtain special records such as electrocardiograms or other laboratory data.

The mainstay in the management of this disease is the reduction to a minimum of the demands on the heart and circulation. To this end the patient should be kept in bed and not be permitted to move about unnecessarily. During the first week or so he should not feed himself. Examinations of the patient, with the exception of auscultation of the anterior chest, should be infrequent during the first few days.

Special nursing care should be instituted immediately. Day and night nurses are essential with special attention to the ability of the night nurse who should be alert and conscientious during her stay on duty. Careful nursing is one of the most important factors in the successful recovery of the patient. No visitors except those in the immediate family should be allowed in the sick-room during the first week. All visits should be curtailed to only a few minutes.

Complete mental relaxation bordering on drowsiness should be maintained during the first few days or even the first week. Highly excitable, apprehensive or temperamental patients are much better off if kept asleep more or less continuously during the early phase of the illness. Some form of a sedative or an opiate may be required to accomplish this. In the absence of pain one of the barbituric acid preparations such as phenobarbital in doses of 30 mgm. (½ gr.) or amytal 0.1 gm. (1½ gr.) given three times a day may be sufficient to keep the patient at ease, drowsy or even asleep. Chloral hydrate or sodium bromide or both may also be given several times daily with good results.

Before leaving this part of the treatment, let me remind you that alcohol is of distinct benefit in the therapy of myocardial infarction. It induces in most individuals peripheral and possibly coronary vasodilatation. Incidentally, Heberden demonstrated long ago that alcohol might not only abort but even prevent angina pectoris in many instances if employed in timely doses. In many patients alcohol produces tranquility and relaxation as well as creating a sense of well-being. To obtain the maximum good effect from its use, alcohol should be administered in that form agreeable to the patient. This is often decided by individual taste or preference although occasionally by race and custom. Consider the likes and dislikes of the patient, and do not give it to those who dislike it in any form. Needless to say do not continue to use it when it fails to be of benefit. Discontinue it if the patient becomes depressed under its influence or if it causes gastric hyperacidity or heart-burn. More often, however, some form of alcohol offsets the depression incident to myocardial infarction and its prolonged period of bedrest and disability.

Small doses such as 15 to 30 cc. (½ to 1 oz.) repeated two or three times a day are preferable to larger amounts given once a day. Let me also stress the importance of not giving alcoholic beverages with large amounts of iced, charged water. Whiskey can be given with plain tap water or small amounts of a natural effervescent water; rum in hot, weak tea is excellent and brandy or sherry may be taken straight or in a frappé. Another pleasant drink and food is bourbon whiskey served in an egg-nog made with milk instead of cream. One of the most pleasant effects of alcohol is its sedative and hypnotic action. A glass of wine, sherry or beer, or a "hot toddy" will often prepare the patient for sound sleep. This will negate the need for drug administration and may result

in a night of normal sleep.

Coffee should be prohibited at least during the first week and preferably for the entire illness. It does no good, despite the fact that caffeine is supposed to cause coronary dilatation;[8] causes precordial pain; and not infrequently does harm by increasing the nervous tension of the patient or instigating the production of premature systoles.

Tobacco of any type should be stopped as soon as the diagnosis of myocardial infarction has been made and the patient should discontinue smoking for at least the remainder of the illness. It is harmful and may actually induce attacks of angina in a goodly proportion of patients with this disease. What the underlying mechanism in the heart may be cannot be definitely stated but, judging by the recent report of Wilson and Johnston,[9] it is quite likely to be one of vasoconstriction of the coronary arteries. It would seem, therefore, to be a wise policy to curtail its use at least during the period of bed confinement, although it would also seem the part of wisdom for anyone who has suffered a coronary occlusion to abstain permanently from it.

Diet: A low calorie diet is essential in decreasing the metabolic rate and in turn reducing the demands on the heart. It also prevents the patient from gaining excessive weight during the long period of bed confinement. If the patient is obese the use of a low calorie diet is definitely indicated since the greater the weight, the greater is the work load of the heart.

The diet should be liquid during the first few days with milk or milk with vichy, and warm broths, as the main constituents. These should be given approximately every three hours. Water should also be administered but total fluid intake should not exceed 1500 cc. in 24 hours, particularly if there are any suspicious signs of a failing heart. The Karell diet (200 cc. of milk given every four hours) is often employed during the first few days. Many patients are unable to tolerate the iced fruit juices that are so often given during the early phase of this illness. In fact iced drinks in general had better be avoided. Junket, custard, warm cooked cereal and some of the stewed fruits such as apple sauce and puréed vegetables may be added toward the end of the first week. If the patient is unable to take fluids or nourishment by mouth, particularly if vomiting is present, 50 cc. of 25 per cent glucose solution may be given intravenously twice daily together with 5 to 10 per cent glucose solution by rectum through a Harris drip. Large volumes of fluid,

especially infusions, are contraindicated because of the danger of pulmonary edema. If actual dehydration is present hypodermoclysis may be employed or rectal instillations may be given by catheter or Harris drip, usually 5 to 10 per cent solution of glucose in saline.

During the second and third weeks a more complete diet may gradually be resumed to include some solid foods such as chicken, fish, lamb chops, cooked vegetables without too much roughage, or which do not provoke flatulence, and some of the raw but readily digested fruits. An adequate supply of vitamins should be provided especially during the first week when food intake is restricted. Deficiency of Vitamin B_1 can lead to heart failure or aggravate the degree of failure which may be instigated by the myocardial infarct.

Bowel hygiene: Regulation of bowel function is essential but may be neglected, as a rule, during the first two or three days, particularly if food intake has been limited to fluids. Straining at defecation should be avoided by keeping the stool soft. This may be accomplished by giving sufficient fluids by mouth and the regular administration of 30 to 45 cc. (1 to 1½ oz.) of mineral oil every night followed in the morning by a small enema of oil and glycerine or saline. After the second or third day enemas may be given daily or every second day, if needed, for the next week or so. Subsequently mild catharsis may be stimulated by the use of milk of magnesia or any other mild laxative.

Abdominal distention is sometimes a problem in the early stage of myocardial infarction. Heat applied to the abdomen may give some relief, also the insertion of a small rectal tube. If not successful small turpentine enemas given with care may be employed. Pitressin[10] may be given but with caution since it is a coronary vasoconstrictor. The newer synthetic forms of atropine such as Novatropine or Trasentin are sometimes effective, particularly if distention is secondary to a spastic colon or some other neurogenic state. Small doses of fluid cascara given repeatedly will sometimes relieve distention.

Some modification in bowel hygiene may have to be made after the first week or so for those patients, usually men, who struggle with the bed-pan. A compromise may be made by allowing the use of a commode placed immediately adjacent to the bed onto which the patient may be lifted or slid by means of a special draw-sheet. Needless to say it is to be used only for bowel evacuation.

Management of immediate attack: Control of the severe substernal

pain or distress which introduces most of the attacks of myocardial infarction is highly essential. Nitrites are ineffective. In fact they may do harm and therefore should not be employed. They are especially dangerous if the patient is in shock. Morphine sulphate given subcutaneously in doses of 15 mgm. (¼ gr.) and repeated several times in the space of an hour or two if needed should be administered. Occasionally it fails to provide relief. Although it appears to be the drug of choice in quieting the patient and relieving pain during the period of severe distress and anxiety, it might be well not to repeat it too often since it not infrequently causes vomiting. If the patient is known to be sensitive to this drug Pantopon 20 mgm. (1/3 gr.) or Dilaudid 4 mgm. (1/12 gr.) may be used hypodermatically.

It may be appropriate here to mention the newer synthetic morphine substitutes which are at present only available for investigation purposes but which will probably be released to the profession in the near future. One of the most promising is known as "Metopon"[11] which is a methyl derivative of dilaudid. Its advantages over morphine include less respiratory depression, greater analgesic potency, less liability for producing addiction, and less noticeable emetic properties. Another one is "Demoral" which has recently been investigated clinically at Bellevue Hospital by Batterman.[12] It is a synthetic analgesic approaching the potency of morphine for the relief of pain. Although having a shorter duration of action, it has the advantage over the opiates in not resulting in respiratory or profound cerebral depression. Prolonged use with therapeutic doses has not resulted in primary addiction. Both these new drugs may some day be very useful adjuncts or may even replace the present day narcotics in the treatment of myocardial infarction.

Recently another drug has been advocated during the early phase of myocardial infarction. I refer to atropine sulphate which is supposed to counteract the efferent vagus action which induces reflex constriction in coronary vessels at the onset of attacks of coronary thrombosis. Experimental studies[13,14] suggest that this action of the vagus may aggravate the cardiac lesion already present and may even precipitate sudden death, probably by initiating ventricular fibrillation. Hence the use of atropine given intravenously in doses of 0.8 mgm. (1/75 gr.) which may be repeated several times daily during the acute stage particularly if pain continues. In the presence of a tachycardia, however, it should be withheld or given in distinctly smaller doses.

The experiments of Manning, McEachern and Hall[14,15] demonstrated that the ligation of a coronary artery of the dog causes a reflex vasoconstriction of the other coronary vessels. When a coronary artery was ligated under complete anesthesia, there was a low mortality, but if the ligatures were not tied until recovery from anesthesia, there was a high mortality. However, if the vagi were cut or atropine administered before the artery was ligated in the unanesthetized dog, the mortality remained at the same low level as when the artery was tied in the animal in whom the reflexes were abolished by complete anesthesia.

A year ago, during the Graduate Fortnight, Gilbert[16] in discussing "the influence of extrinsic factors on the coronary flow and clinical course of heart disease," referred to some unpublished work, subsequently reported,[13] which showed that the administration of a purine-base vasodilator, such as aminophylline, in addition to atropine, caused an even lower mortality when the coronary artery of a dog is ligated. They have already made use of these observations in the management of coronary occlusion in humans. By the immediate administration of atropine and aminophylline they believe that they have not only greatly reduced their mortality in early cases of coronary thrombosis but the recovery of their patients has been more rapid and more uneventful than previously.

Some authorities[17,18] use aminophylline alone, others[16] combine it with atropine or papaverine but both administer it intravenously in doses ranging from 0.24 to 0.48 gm. two to three times a day during the acute or critical phase of myocardial infarction. Both groups feel that the results obtained with aminophylline are due to an improvement of the collateral coronary circulation. My own experience with the intravenous use of aminophylline in myocardial infarction, either given alone or with atropine or papaverine, has been rather limited. I have seen satisfactory results in some cases, equivocal or even poor results in others. Although I believe these drugs are valuable in the treatment of an acute myocardial infarct I feel that administration intravenously should be restricted to the critically ill patients rather than given routinely.

If not obtainable in the 10 or 20 cc. ampule, aminophylline should be diluted to this amount with physiological saline, dextrose solution, or distilled water. Injection should be very slow and preferably through a fine gauge needle (22 gauge). Rapid injection may cause fulness in the

head or a throbbing headache, vertigo, palpitation, and even precordial pain or oppression. Occasional fainting may take place, especially in sensitive or neurotic patients.[18,19] Smaller doses such as 0.24 gm. (3¾ gr.) should be used in those patients with accelerated heart rates or whose blood pressure is very low, since the drug sometimes causes a fall in blood pressure and/or a tachycardia.

In some patients complete relief of substernal pain or distress is noted almost immediately, even in the absence of an opiate. Reduction in the amount of opiates administered is often possible. It sometimes may be withheld completely when aminophylline or atropine or papaverine are used. However, the latter drugs should not be employed to the exclusion of other important therapeutic measures and should be used with caution and discontinued with the appearance of severe reactions.

Aminophylline may also be given by mouth, preferably in the form of an enteric-coated capsule or tablet to avoid or reduce the gastric irritation which so frequently occurs in those individuals sensitive to the drug or in those patients in whom large doses have been employed. These symptoms include nausea or vomiting, burning epigastric pain, and distention. It may be given orally in doses of 0.2 gm. (3 gr.) three or four times a day for variable periods of time during the first month or two after the onset of the illness and subsequent to the intravenous use of the drug during the critical phase. Gastric irritation may be avoided by discontinuing the drug three to four days at a time, and then resuming for a week or ten days.

There is still considerable difference of opinion concerning the value of aminophylline as a coronary vasodilator both in animals and in man, and especially in the presence of coronary sclerosis or occlusion. Experimental studies in animals[20,21,22] have given variable, even contradictory, results. In man, however, Levy, et al.,[23] using induced anoxemia in individuals subject to attacks of angina caused by coronary sclerosis, found that aminophylline caused a prolongation of 63 per cent in the time of appearance of pain when given intravenously but only 26 per cent when taken by mouth. Clinical tests[24,25] controlled by placebos have shown that aminophylline or other xanthines administered orally have no superiority over the placebos. Others have[26,27] obtained good results with these drugs but employing considerably larger doses. Although the drug is not dangerous when given orally, its intravenous administration is known to have resulted in many instances of reactions, previously men-

tioned, which may be rather disturbing.[18] A few fatalities have been noted but not reported as far as I am able to ascertain. One observer[28] believes that he induced an attack of coronary occlusion in two patients with bronchial asthma—one associated with a previous myocardial infarct, the other probably with coronary sclerosis—by using 0.48 gm. of aminophylline intravenously. One of these patients succumbed three months later but no autopsy was obtained.

Fortunately, an appreciable number of patients follow a rather benign course from the onset of their myocardial infarct through to convalescence. Herrick referred briefly to these cases in his first communication in 1912. In these patients the pain not infrequently disappears shortly after onset or even before the physician's arrival and no other severe manifestations of the disease such as shock, embolization, or heart failure make their appearance. Needless to say, in such instances therapeutic measures can be quite limited and intravenous aminophylline or atropine or both are probably of doubtful value or even contraindicated. Overtreatment of the patient in such instances is more to be feared than neglect.

Papaverine: Although not a new preparation, this drug has had somewhat of a renascence, so to speak, in the past few years in the treatment of all forms of vascular disease. It is an opium alkaloid of low toxicity and non-habit forming. It has been shown to be very useful in coronary occlusion, chiefly because it is a powerful coronary vasodilator.[29] Experiments on dogs by McEachern and his Toronto group[30] show that the administration of this drug reduces the mortality resulting from ligation of a major coronary artery. Likewise, the experimental demonstration by Katz, et al.[31 32] that this agent reduces or abolishes induced premature contractions and decreases the ease with which ventricular fibrillation is induced in the dog, suggests the possibility of its clinical value in the treatment of premature systoles, and its use in conditions which are apt to lead to ventricular fibrillation.

As to its mode of action it has been credited by Katz[32] with the following effects: A mild sedative action, a definite and lasting coronary dilating action, and the prevention or lessening of the occurrence of premature systoles (auricular, nodal and ventricular) or certain types of rapid heart action like ventricular fibrillation. In this last respect it acts like, but apparently is superior to, quinidine, since it also has a coronary dilating action which quinidine lacks. Furthermore, it is not a

myocardial depressant like the latter drug.

Papaverine, therefore, may be of considerable value in fresh myocardial infarction since premature systoles occurring in this process represent an added burden on the heart and may, if of ventricular origin, result in ventricular tachycardia or fibrillation. Relief of pain in this disease has also been obtained by its intravenous use, employing it in doses of 60 to 90 mgm. (1 to 1½ gr.), repeated every two to three hours if necessary. Oral administration can be used to continue its desired effect, the usual dose then being 0.2 gm. (3 gr.) every three to four hours.

Oxygen therapy: In acute coronary occlusion a sudden interference with the flow of blood results in a severe oxygen-want in the myocardium. The use of oxygen in this disease was first suggested in 1929. In the following year and again in 1934 Levy and Barach[33] demonstrated its value as a therapeutic agent in this disease. According to these authorities[34] the basis of its use is founded on three facts: (1) "Anoxemia of the heart muscle occurs after sudden occlusion of a sizeable branch; (2) oxygen-want induces impairment of cardiac and respiratory activity; (3) the inhalation of oxygen in high concentration increases the oxygen content of the arterial blood and results in improvement in the functional capacity of the heart."

Oxygen administration in high concentration,[35] even in 100 per cent concentration for periods of approximately 12 hours, is particularly indicated in the patient with myocardial infarction who presents circulatory collapse, or severe pain and restlessness which does not respond to the opiates or other medications, and when there are evidences of a failing myocardium. The latter may be suggested by the appearance of cyanosis, tachycardia, gallop rhythm, low blood pressure, dyspnea, and signs of pulmonary congestion. It is also very effective in the treatment of pulmonary edema as a result of left ventricular failure, particularly if given by the positive pressure technique.[36] Paroxysmal dyspnea and Cheyne-Stokes breathing also respond to oxygen therapy.

Oxygen tents are capable of delivering and maintaining from 50 to 75 per cent of oxygen. They are comfortable to most patients. The oronasal catheter is simple and efficient but must be carefully placed under direct throat vision. It is said to deliver from 35 to 50 per cent of oxygen. Recently various types of masks have made their appearance including the well-known Boothby mask, both nasal and oronasal types,

which are capable of delivering up to 100 per cent oxygen. They are quite simple, efficient, inexpensive and easily adjusted. However, many patients complain that they are uncomfortable, especially in warm weather. Small face tents are also obtainable, made of transparent plastocele, which fit over the bridge of the nose and may easily be molded to the conformation of the patient's face. This may give oxygen concentration between 40 and 60 per cent. Many patients find this type more comfortable, especially in warm weather than the heavier rubber masks or the nasal catheters.

Complications: More than 50 per cent of the attacks of myocardial infarction are associated with complications and the majority of these occur during the first three weeks. A fairly common one is that of peripheral circulatory failure of some degree which invariably occurs with or immediately after the onset of the attack.

Acute circulatory failure or shock occurs in as high as 1/3 of cases as a complication of the immediate attack. It may introduce the picture in some instances, with pain as a minor symptom or entirely absent, but more often appears simultaneously with the pain. In other instances it occurs hours or days after the onset of pain. Its manifestations usually constitute a classical picture with cold clammy skin, rapid and shallow respirations with frequent sighing, gray, cyanotic pallor of the face which may be drawn and anxious, rapid, feeble pulse and depressed arterial pressure with small pulse pressure. Although a subnormal mouth temperature may be obtained, the rectal temperature is frequently elevated. The cause of this primary form of shock in coronary occlusion remains obscure. Some form of nervous mechanism seems a likely probability. Acute cardiac weakness and failure of the central driving force are undoubtedly contributing factors in the mechanism. A definite decrease in circulating blood volume has been demonstrated.[37] This may be the result of stasis in the vascular bed and an increase in capillary permeability secondary to tissue anoxia, and in turn resultant loss of plasma and other blood elements.

If the severe substernal pain which introduces the attack of myocardial infarction is not present during shock, morphine should not be administered unless anxiety and restlessness are prominent because it depresses pulmonary as well as tissue respiration and thereby aggravates the general anoxic state already present. High concentration of oxygen, 75 to 100 per cent, administered by one of the special face

masks or by tent should be instituted. Theoretically, vasoconstrictor drugs such as epinephrine should be of some benefit in the shock of myocardial infarction. However, only in rare instances do they seem to be of help. They should be used with caution and preferably in small doses such as 0.5 mgm. subcutaneously or intramuscularly, or 0.1 to 0.2 mgm. of a 1:1000 solution intravenously. Occasionally it is employed much diluted in an intravenous saline drip. It is well known that epinephrine administered to patients with coronary sclerosis readily initiates the anginal syndrome. Hence the caution with which it should be used for shock in coronary occlusion.

I have seen patients with myocardial infarction in shock respond to transfusions of blood or plasma. Such transfusions should be given in quantities ranging from 150 to 250 cc. They probably help by augmenting the decreased circulating blood volume. However, such transfusions should be given slowly in order to prevent overloading the already injured heart. Hypertonic glucose solutions by intravenous infusion may be given slowly in amounts of 100 cc. of a 50 per cent solution and repeated if needed. This may not only increase the circulating volume but also act as nourishment for the heart muscle. It also tends to increase venous return from the extremities to the heart. The intravenous administration of fluids may be hazardous and therefore should be employed with caution. The presence of any degree of heart failure, as determined by clinical or laboratory means (increased venous pressure), is an absolute contraindication.

Elevation of the foot of the bed should be done immediately. The patient should be covered with blankets but artificial heating methods should be withheld since recently some question as to the benefit of artificial heat has been raised. Burns may readily result because the constricted peripheral vessels cannot carry off the excess heat. Then too, if vasodilatation should occur, the blood pressure level will drop further. In addition, excessive sweating may cause further loss of body fluid. Bandaging the lower extremities from ankles to mid-thigh may also be of value. It has been shown that the circulating blood volume may be increased 1000 cc. by this procedure.[38]

Arrhythmias: Various forms of arrhythmias make up the second commonest complication in myocardial infarction. Some, such as complete heart block and ventricular tachycardia, are quite rare; others, such as premature systoles and paroxysmal auricular fibrillation, are more

common. Probably, most sudden deaths which occur during or after the immediate attack or in the first three weeks are due to ventricular fibrillation. Rupture of the heart is a much less frequent cause of sudden death.

Premature systoles, particularly of ventricular origin, are quite common during the early stage. Those occurring infrequently may be ignored unless annoying to the patient. They can usually be abolished by the use of quinidine in doses of 0.2 gm. (3gr.) three times a day or papaverine 0.2 gm. (3 gr.) every four hours. Either one of these drugs should definitely be used if the premature systoles occur frequently, i.e., once in every ten beats or in series, which suggests that they may be the forerunner of ventricular tachycardia. The latter rhythm has been shown to be the link which connects ventricular premature systoles with ventricular fibrillation.[39]

I see no justification in the routine administration of quinidine, as has been advocated, during the first two weeks as a prophylactic against the development of ventricular tachycardia or fibrillation. Both disturbances are quite uncommon in patients who survive the first 24 hours, which the majority do. Their onset is commonly preceded by frequent ventricular premature systoles which act as a warning and thereby permit the institution of corrective measures. Ventricular tachycardia is usually amenable to treatment. Also the efficiency of this measure has never been proven and seems rather difficult of proof. Lastly, since quinidine is a distinct myocardial depressant it seems best to employ it only when actually needed.

Ventricular tachycardia is always of serious significance since it may precipitate heart failure, embolism, ventricular fibrillation and sudden death. Although its recognition is only certain by means of the electrocardiogram, its presence should be suspected in any patient with myocardial infarction who suddenly exhibits a rapid rate ranging from 180 to 200 per minute which is basically regular in rhythm. Fortunately, the paroxysm may end spontaneously within a short period after onset. Others, however, continue for hours or even days.

Several drugs are known to influence this rhythm. They include quinidine salts, magnesium sulphate, and potassium chloride or acetate. When quinidine is to be administered, it is well to give a preliminary test dose of 0.2 gm. (3 gr.) by mouth and wait 30 minutes or so to note if any signs of sensitivity to the drug such as marked tinnitus, diarrhea

or vertigo appear. If not, then doses of 0.4 gm. or even 1 gm. (6 to 15 gr.) may be given at intervals of two or three hours until the bout terminates or signs of toxicity appear. Following return to normal, quinidine in doses of 0.2 gm. (3 gr.) three times daily after meals should be continued for the next week or two to diminish the possibility of recurrence.

Magnesium sulphate may be given intravenously in a 10 to 20 per cent solution in amounts up to 15 cc. It should be injected very slowly, pausing after every 5 cc. to check the heart rate since the attack may stop before the entire amount is injected. It acts by depressing the ectopic focus which is usually the infarct or muscle immediately adjacent to it.

Potassium salts are known to depress the conductivity and excitability of the myocardium. They are also known to potentiate digitalis and quinidine, and finally, when given alone, may abolish premature systoles and paroxysmal ventricular tachycardia.[40] Either potassium chloride or acetate[41] may be employed, using 1 to 2 gm. (15 to 30 gr.) every two to four hours by mouth, until a favorable response is obtained or a maximum of 10 gm. (150 gr.) has been administered.

Another procedure which may be tried, particularly if quinidine or the potassium salts alone are ineffective, is the administration of both drugs.[40] Quinidine is then given in full doses until 2 to 3 gm. (30 to 45 gr.) have been administered, at which time the potassium salts should be started.

Formerly quinine dihydrochloride was used intravenously to abolish prolonged attacks of ventricular tachycardia and more recently quinidine sulphate. However, the intravenous use of either drug is dangerous because of their severe reaction on the myocardium. Sudden death may occur with both and several such incidents have been reported. Only when all other measures have failed and the outlook appears hopeless would I suggest the intravenous administration of these drugs.

Paroxysmal auricular fibrillation occurred in 12 per cent of an unpublished series[2] of 200 cases with myocardial infarction at Bellevue Hospital. The majority of attacks are of short duration ending spontaneously. Some, however, continue for days or the rhythm may become established. It is the longer episodes which, as a rule, need treatment since they may, and not infrequently do, precipitate heart failure. The drug of choice in such instances is digitalis given in divided

doses and always by mouth. An initial dose of 0.4 gm. (6 gr.) may be given followed by 0.4 gm. (6 gr.) at intervals of six hours until normal rhythm returns or the ventricular rate reaches 70 or 80 per minute. If sinus rhythm is not obtained by digitalis, quinidine in doses of 0.3 gm. (5 gr.) at intervals of two hours may be employed. If not successful alone quinidine may be supplemented with strychnine sulphate 1.5 mgm. (1/40 gr.) three times a day. This combination has been shown by Smith and Boland[42] of the Mayo Clinic to be more efficacious in auricular fibrillation than when quinidine is administered alone and smaller doses of quinidine are required if strychnine is also administered. Should fibrillation persist despite quinidine therapy, digitalis may be resumed in maintenance doses of 0.1 gm. or 0.2 gm. once daily.

Congestive heart failure in our experience rarely occurs to any obvious degree in individuals sustaining their first attack of myocardial infarction unless they are 60 years or over, or have a complicating disease such as diabetes mellitus or hypertension.

The clinical signs of congestive failure may not be too obvious as judged by the usual manifestations of dyspnea, pulmonary rales, edema and engorged liver. Venous pressure determinations, however, are frequently elevated even in the absence of symptoms or when only a few basal rales may be the only indication of a failing heart.

The hypertensive cardiac who sustains a myocardial infarct, in contrast to the uncomplicated arteriosclerotic cardiac, quite often develops paroxysmal dyspnea as the prominent clinical phenomenon of acute left ventricular failure. Several therapeutic measures seem effective in this condition. First of all, morphine hypodermatically in a 15 mgm. (¼ gr.) dose should be given immediately. If available, administration of high concentration of oxygen by tent or mask is of value, and if pulmonary edema complicates the picture, it may be given under positive pressure.[36]

Venesection or phlebotomy may successfully be carried out by the removal of 500 cc. of blood. However, an effect similar to this may be obtained by applying blood pressure cuffs to the extremities and inflating them to a pressure slightly greater than the patient's diastolic pressure. Dramatic relief may be obtained by this means. The purpose is to utilize the peripheral venous system as a reservoir and thereby decrease the circulating blood volume. It has one drawback, it causes venous dilatation and thus may favor venous thrombosis and possibly emboli.

An instrument has recently been devised by Kountz et al.[43] which inflates and deflates cuffs, placed on the arms and thighs, in rotation. They believe that as much as an eighth of the total circulating blood volume may be impounded by their method of intermittent constriction. Their application of this procedure in patients with acute left ventricular failure usually resulted in dramatic improvement. They also suggest that acute myocardial infarction is another condition in which reduction of the quantity of blood-flow to the heart might be beneficial in minimizing the demands on the myocardium. In 10 patients with acute myocardial infarction this treatment was given for a period of 14 days. All of them survived, whereas the mortality rate of such patients in a hospital from which a similar group of patients was chosen, was 37 per cent. They state that although a more detailed study must be made before definite conclusions can be drawn, the results suggest that this treatment affords some protection to the heart when the function of the left ventricle is impaired.

Aminophylline, given intravenously in doses of 0.48 gm. (7½ gr.) in 10 or 20 cc. of saline or hypertonic glucose solution, is another drug which is quite effective in acute left ventricular failure. Concentrated glucose solution, such as 50 cc. of a 50 per cent solution, has also been employed alone, particularly when pulmonary edema is present, with seemingly good results.

Mercurial diuretics are of distinct value in acute failure although their full therapeutic action is delayed longer than the preparations and procedures just mentioned. However, because of the recent "wave" of reports in the medical literature[44,45] of instances of sudden death or severe reaction following their administration, it might be well to stress a few notes of caution:

1. Never use more than 2 cc. for the initial intravenous dose. A preliminary dose of 1 cc. intravenously would be preferable for the patient with myocardial infarction.

2. Always draw blood into the syringe before injecting the drug and take plenty of time to inject, employing a small calibre needle (25 gauge).

3. Check the patient for evidence of salt depletion. If present give sodium chloride for one or two days prior to the administration of the diuretic.

4. If the patient has been taking digitalis, be certain that the digitalis

dosage is well below the toxic level, or discontinue it for a day or two. It might be well also to use a small dose, i.e., 0.5 cc. to 1 cc., at the initial injection intravenously. Larger doses can be given later if satisfactory diuresis does not result.

Mercurial diuretics may be used rectally in suppository form; more recently a tablet has been offered for oral use. Neither one is as effective as the intravenous or intramuscular preparations. All forms of mercurial diuretics are made more effective by the administration by mouth of ammonium chloride, ammonium nitrate or potassium chloride, preferably given in enteric-coated capsules.

Although it is of distinct importance to call attention to the dangers of these diuretics it seems just as important not to exaggerate these dangers since these preparations constitute one of the most valuable groups of therapeutic agents in the treatment of diseases of the heart. As DeGraff[46] has stated, one must remember that the toxic reactions and deaths reported from mercurial diuretics are in reality only a very small number in relation to the extensive use of these drugs. These reports, therefore, should not discourage the rational use of these diuretics.[47]

Digitalis. I have purposely left this drug to the last because of its disputed status in myocardial infarction. As a rule digitalis is not needed in the immediate treatment of paroxysmal dyspnea with or without edema since the therapy just outlined is usually effective. However, since this mechanism is indicative of left ventricular failure it may be desirable in some instances to follow the administration of the rapidly acting drugs by digitalis with the hope of maintaining the patient's cardiac reserve.

Indications for the use of digitalis in myocardial infarction include (1) the appearance of progressive congestive failure, either with or without preceding left ventricular failure, which has not responded to the administration of mercurial diuretics, fluid intake restrictions, sedation, or high concentration of oxygen, and (2) the control of paroxysmal auricular fibrillation, particularly the attack with a rapid ventricular rate or that which is complicated by signs of congestive failure.

I would suggest that digitalis should always be given in such cases by mouth or by rectum and never intravenously. Dosage should be smaller than in the average cardiac and given more cautiously with care-

ful supervision during administration and frequent check-up for early therapeutic effect or toxic signs or symptoms. I would advise the general practitioner to use the tablet of U.S.P. powdered digitalis leaf in the patient with myocardial infarction rather than any of the new digitalis glycosides, at least for the present. Goodman[11] of New Haven pointed out in a recent lecture at the Academy that in the overwhelming majority of patients with heart failure one will find this preparation easy to administer, readily absorbed when given by mouth, effective in its action on the myocardium; also that it possesses an adequate amount of safety, is stable in potency, and cumulation and elimination occur in the body at rates allowing complete digitalization at varying speeds and satisfactory maintenance of desired therapeutic effects for long periods of time. Undoubtedly Doctor Gold will discuss this problem in detail in a subsequent lecture.

The fear of cardiac rupture in myocardial infarction due to digitalis therapy has been highly exaggerated. It may interest you to know that in a large series of myocardial infarcts with rupture reported from the Pathological Laboratory of Bellevue Hospital,[48] none of the individuals had received digitalis.

The other fear has more to substantiate it, namely, to instigate an arrhythmia due to the irritability of the injured myocardium. I believe this is a likely relationship although once again let me remind you of the appreciable number of patients who spontaneously, without digitalis administration, develop rhythmic disturbances including ventricular tachycardia and ventricular fibrillation. Therefore, I again repeat that if the drug is indicated it should be administered but with caution and careful supervision.

Embolism. This occurs in 12 per cent of cases of myocardial infarction.[2] It usually takes place during the first six or eight weeks. The source of the emboli is usually the mural thrombus in the left ventricle which commonly forms on the endocardial surface of the infarct. Some day it may be demonstrated that this will be prevented by the use of an anticoagulant such as heparin or dicoumarin. Emboli may lodge in any part of the systemic or pulmonary circulation, depending on the site of origin, but the brain, the peripheral arteries of the extremities, and the lungs are the most important sites. Pulmonary emboli are apt to occur later, after the third week, since their source is usually not in the heart but in the veins of the pelvis or lower extremities. Here throm-

bus formation is initiated by the prolonged inactivity of the patient and other concomitant factors. Large emboli breaking off from these sites are occasionally the cause of sudden death during the course of myocardial infarction.

The treatment of choice includes immediate intravenous papaverine in large doses, 0.1 to 0.2 gm. (1½ to 3 gr.), repeated in two hours if needed.[49,50,51] This drug may bring about a rapid return of color and function to the affected extremity accompanied by disappearance of pain. It may also be useful in cerebral embolism and very definitely so for pulmonary emboli. When the extremities are involved, paravertebral sympathetic block is another beneficial measure in some cases in reducing vasconstriction of collateral vessels as well as in major vessels. Embolectomy is probably too dangerous in the presence of fresh myocardial infarction because of the bad operative risk in these patients. However, Pratt[52] believes that embolectomy is indicated in every instance when the diagnosis is made, even though an occasional patient will survive without the removal of the embolus.

Diabetes Mellitus. That coronary occlusion with myocardial infarction is not an infrequent occurrence in the patient with diabetes mellitus is common knowledge. It also is generally known that caution must be exercised in the use of insulin in this combination of diseases. Although glycosuria and hyperglycemia may be treated conservatively in such instances, acidosis cannot, since it may cause death. Nevertheless, some[53] feel that insulin should not be used in the presence of coronary thrombosis since it is said to place an added burden upon the heart and may cause hypoglycemic shock, acute coronary insufficiency, or even death. Having seen several patients with myocardial infarction needlessly go into diabetic coma because insulin was withheld, I am rather adamant in stating that this drug should not be withheld, if needed, even when diabetes is complicated by coronary occlusion. Obviously both the dietary regime and insulin administration in these patients should be modified. This may be done by reducing each individual dose but, if necessary, increasing the number of injections so as to avoid hypoglycemia and yet be sufficient to prevent acidosis.

In some unpublished experiments performed by Ralli,[54] diabetic patients in the arteriosclerotic age period, including some with a diagnosis of coronary sclerosis, were given insulin in divided doses sufficient to prevent hyperglycemia and glycosuria but not enough to cause hypo-

glycemia. Serial electrocardiograms were taken frequently during the course of administration. In none were there any obvious deviations from the normal.

As Joslin[55] has recently emphasized "diabetic coma is an acute deficiency state, a condition in which the primary disturbance is a lack of insulin." He also states that "when a patient with diabetes is seen in diabetic coma it is proof that someone has blundered and, if death due to diabetic coma occurs, the rule still holds." I should like to add that this holds true even when the patient has a complicating coronary occlusion.

It apparently is wiser to use regular insulin in patients with myocardial infarction since severe insulin shock seems more apt to occur with protamine zinc insulin.[56]

In closing I merely wish to say that I have only touched on what I felt were the highlights of the problem assigned to me for discussion.

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DEATHS OF FELLOWS

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Dr. Goldwater was superintendent of the Mount Sinai Hospital, 1903-16, and Director of that institution, 1917-29; Commissioner of Health of New York City, 1914-15; Commissioner of Department of Hospitals of the City of New York, 1934-40; a trustee and, since 1940, president of the Associated Hospital Service of New York. He was consulting expert on hospital construction, Bellevue and allied hospitals, 1908-15; consultant on Health and Hospitals of the City of New York, 1917; consulting expert, United States Public Health Service, 1918, 1930; consultant on Hospital Planning and Construction, United States Veterans' Bureau, 1924; member of the American Hospital Association and its president in 1908; member of the National Institute of Social Sciences and its vice-president, 1918-21; honorary member of the British Hospital Association; president of the American Conference on Hospital Service, 1924-26; vice-president of the Research Council, Department of Hospitals, New York City since 1941; medical counsellor, U. S. Veterans' Bureau, 1924; consultant expert to the Institute of Experimental Medicine at Leningrad, Russia, 1933; consultant to the Murry and Leonie Guggenheim Foundation; consultant to the Pan-American Sanitary Bureau; member of the Committee on Hospital Service of the American Hospital Association, Chairman of the Council on Administrative Practice of the American Hospital Association, and a member of the Advisory Committee to the Council on Health and Hospitals of the American Medical Association.

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